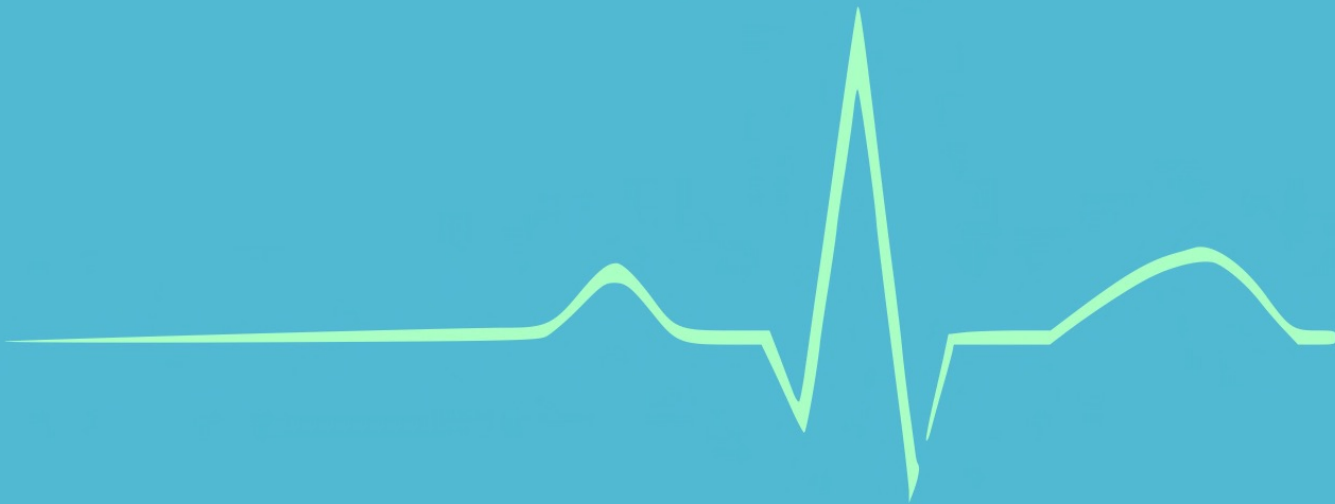
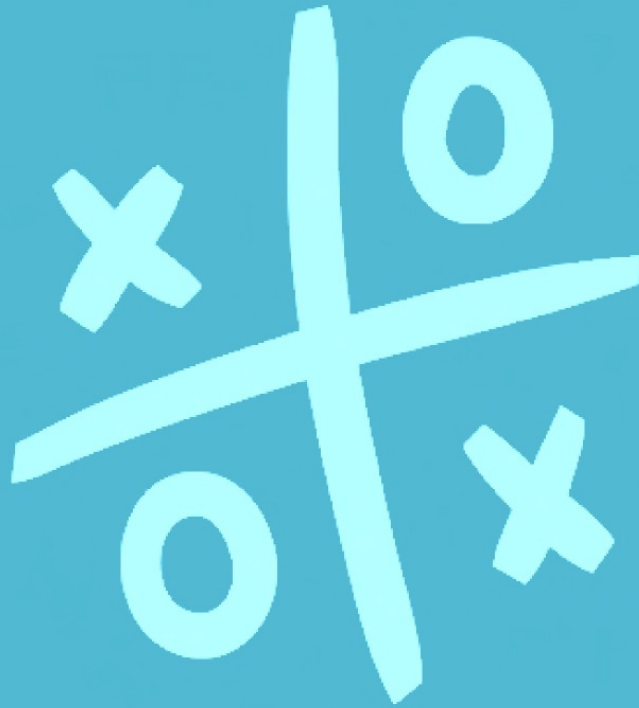


ECG / ABG & Miscellaneous

Based on Dr. Rakesh Nair's Marrow 4.0 Lecture



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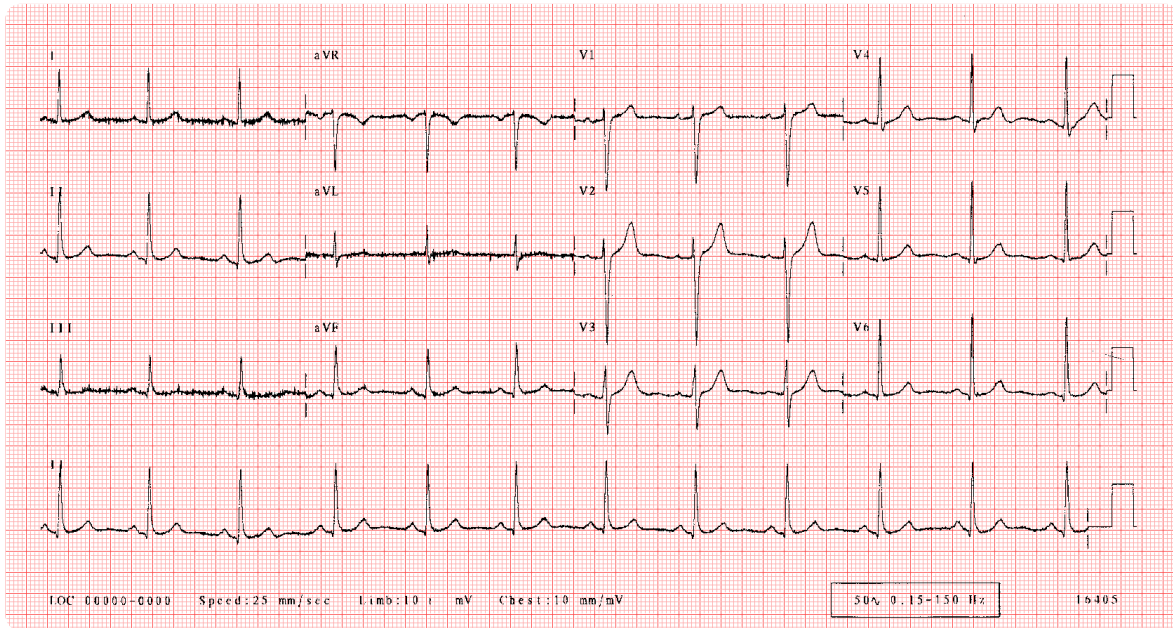
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BASICS OF ECG

CATEGORIES OF ECG

1. Tachyarrhythmias
2. Bradyarrhythmias
3. Localizing MI
4. Miscellaneous

ANALYSIS OF ECG



✓ Whenever the question is looking for an arrhythmia as the answer **focus on RHYTHM STRIP**. (It's always the lead II)

✓ First look on **STEP-1**
QRS Complex

NOTE:
Every small boxes on ECG = $40\text{ms} = 0.04\text{s}$

stands for

VENTRICULAR DEPOLARISATION

↓ occurs in

(A) **SEPTAL DEPOLARISATION**

→ Always proceeds from Lt → Rt
(∵ Lt bundle branch is shorter)
(∵ Rt leads will be positive)

→ V_1 (Rt leads) : small positive R-wave

V_5 - V_6 (Lt leads) : small negative Q-wave

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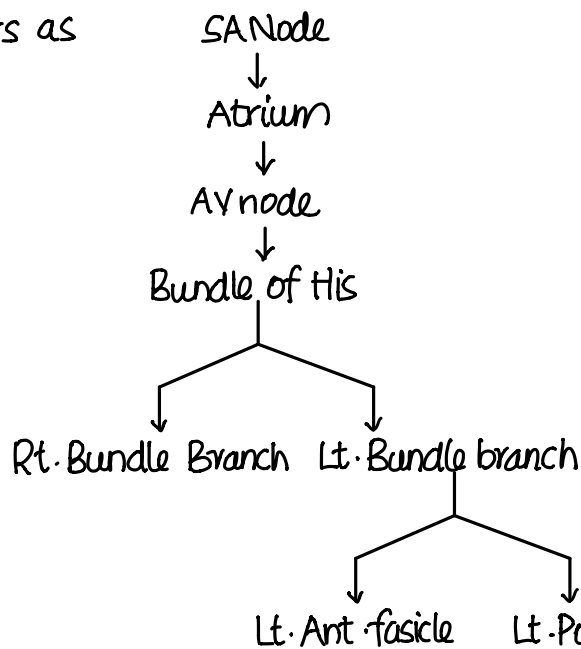
→ Small process ∴ deflection is also minimal



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(B) MYOCARDIAL DEPOLARISATION

→ Occurs as



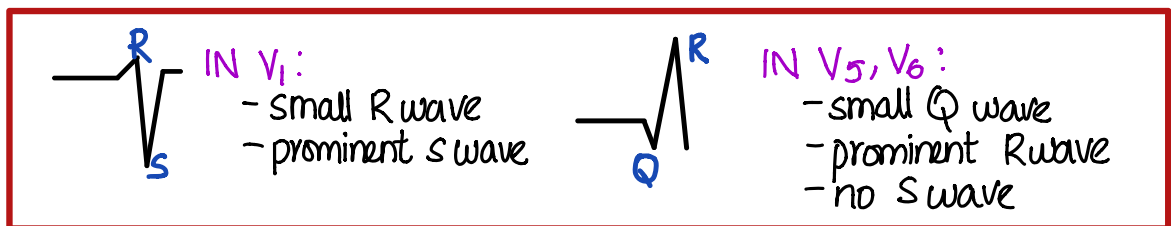
→ Both these synchronously cause Ventricular depolarisation.

FROM ENDOCARDIUM → MYOCARDIUM

Purkinji fibers situated
(∴ conduction is fast)

Bulkiest part

→ Direction is from Rt → Lt (∴ Lt sided leads will be positive)
∴ in V₅-V₆ : Prominent R wave
in V₁ : Prominent S wave



(C) POSTEROBASAL LY & POSTEROBASAL SEPTUM

→ 3rd parts to get depolarised

→ Terminal part of Ventricular depolarisation: APEX to BASE

WIDE QRS COMPLEX

• That means conduction not occurred via Purkinji System

CONDITIONS IN WHICH WIDE QRS COMPLEX PRESENT

(a) Ventricular Pre excitation

- ventricular premature complex (or) Ventricular tachycardia
- Ventricle is actually the source of Rhythm or the focus → WIDE QRS

(b) Aberrant Pathway

- Accessory pathway present → activate ventricle : WPW Syndrome

(C) Bundle Branch block

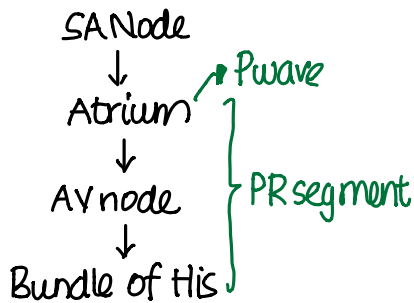
- Either Left or Right

STEP-2 : Check it's Sinus Rhythm or what?

✓ Now check : QRS Complexes are preceded by P wave

i.e Ventricular activation occurred from above

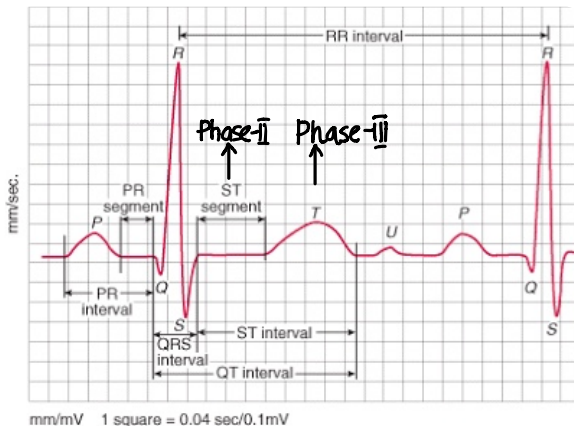
✓ Next Look into : PR interval → 3-5 small boxes = 120-200 ms



Beginning of P wave to beginning of QRS
P wave + PR segment

Denotes the activation of atria

Time taken to impulse to move from Atria to AV node, inside AV node & Upper part of Bundle of his



→ Short PR interval

: < 120 ms = < 3 small boxes

→ Prolonged PR interval

: > 200 ms = > 5 small boxes

Impulse can be originated from

SINUS : Normal Morphology P wave

OR

ATRIUM : Abnormal Morphology P wave

OR

AV NODE : No P wave

OR

VENTRICLE : QRS complex is wide

AV Nodal Delay : Normal phenomenon

- When the impulse travels across AV node it takes some time
- i.e the reason the PR interval is b/w 3-5 small boxes

PROLONGED PR INTERVAL → AV BLOCK

SHORTENED PR INTERVAL → WPW SYNDROME

(Aberrant pathway connects Atria to ventricle bypassing the AV node)

STEP-3 : Check it's Regular or Irregular

✓ Look for : R-R interval is regular or not

STEP-4 : Calculation of Rate

$$\text{Rate} = \frac{1500}{\text{no. of small boxes b/w two R waves}}$$

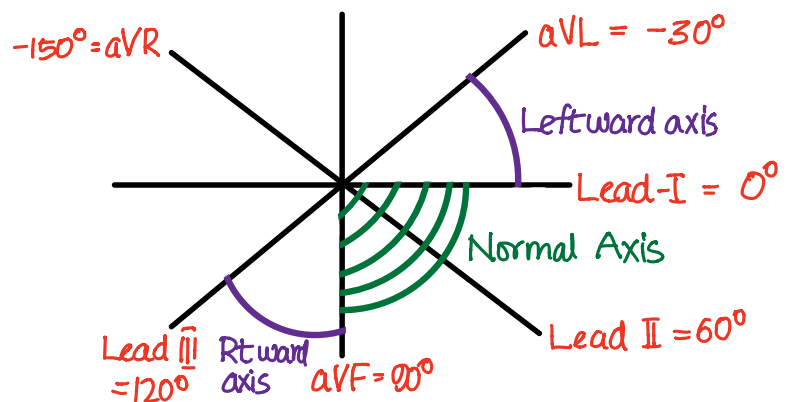
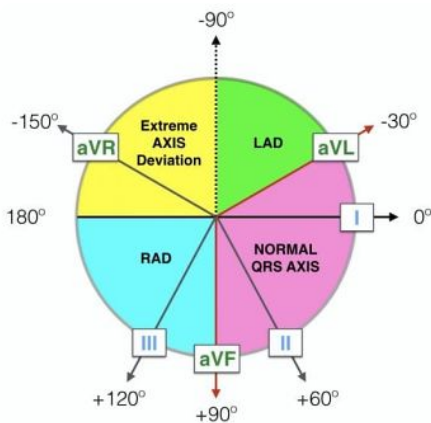
∴ The first ECG is of Normal Sinus Rhythm, Regular with narrow QRS Complex, Rate of 70/minutes.

STEP-5 : Calculation of Axis of ECG

For calculation of axis look into

- Limb leads
- Augmented Limb leads

DONT LOOK INTO CHEST LEADS



- Anything more than -30° = Left Axis Deviation (LAD)
- Anything more than 120° = Right Axis Deviation (RAD)
- Normal axis = $0-90^\circ$
- Leftward axis = $0-(-30^\circ)$
- Rightward axis = $0-120^\circ$

✓ Calculate the voltage of Lead I, II, III, aVL, aVF. (R-wave deflection)

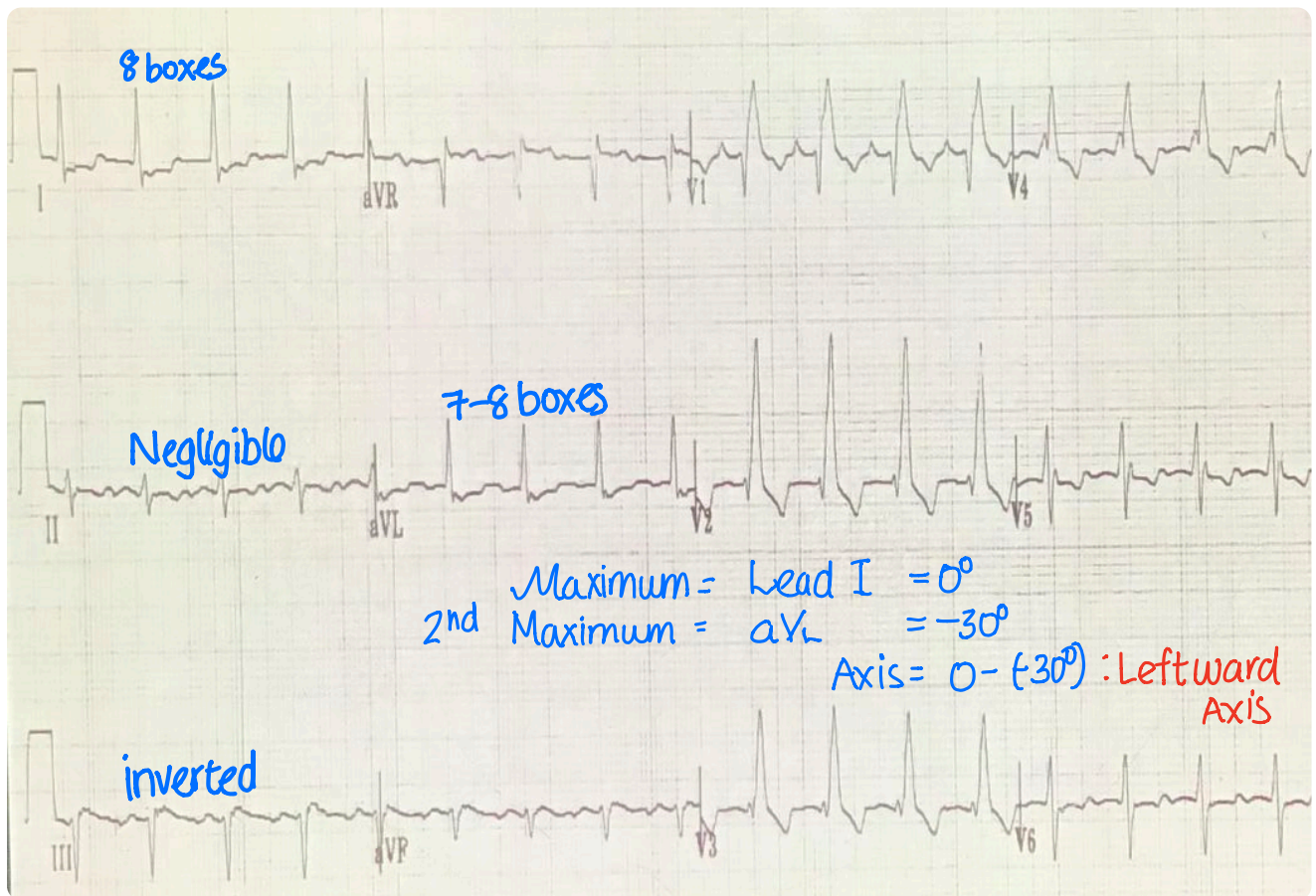
From the 1st ECG: 12 15 8 7 10 small boxes

2nd maximum Max Voltage

0° 60° @neetpgdiscussion

∴ Axis of ECG is b/w 0° & 60° (More likely b/w $30-60^\circ$ as Lead II (60°) is max)

? Calculate the axis of following ECG



STEP-6 : QT Interval

- Some total of Ventricular activity

↓
Ventricular depolarisation (from endocardium to epicardium)

+

Ventricular Repolarisation (from epicardium to endocardium)

- from beginning of QRS complex to end of Twave or U wave

- Normal QT interval = 0.40 - 0.42s = 10-11 small boxes

- Prolong QT interval = >12 small boxes

female is having prolong QT interval → 0.46s ♂
0.48s ♀

$$\text{CORRECTED QT INTERVAL (QTc)} = \frac{QT}{\sqrt{RR}}$$

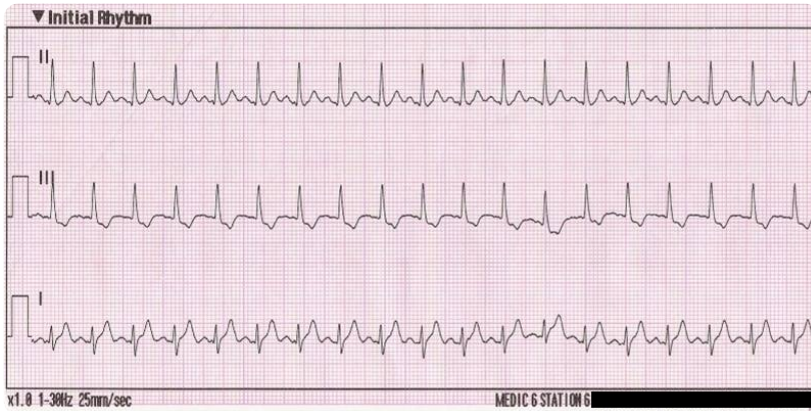
In the First graph : QT = 10 small boxes = 400ms = 0.40s
: RR = 20 small boxes = 800ms = 0.80s

$$QT_c = \frac{0.40}{\sqrt{0.80}}$$



QT Prolongation is due to

- Prolongation of Ventricular Repolarisation
(Phase-III Cardiac A.P Prolongation)



- Concentrate on Rhythm strip

STEP-1 : NARROW QRS
(supraventricular impulse)

STEP-2 : Every QRS preceded by P wave
: PR interval normal

STEP-3 : Regular

STEP-4 : $1500/9 \approx 150$: Tachycardia
Narrow QRS complex Tachycardia.

Sites of origin of Impulse can be

Above it's a Sinus Tachy- : Normal P wave

Abnormal P wave

No P wave

← SINUS → Sinus Tachycardia

or
← ATRIA → Atrial Tachycardia

or
← AV Node → Junctional Tachycardia

- MC Tachycardia
- MC Supraventricular Tachycardia
- MC Narrow QRS Complex Tachycardia

- Rate : < 180 / minutes

if it's > 180 / minutes sinus will not be able to control the impulse.

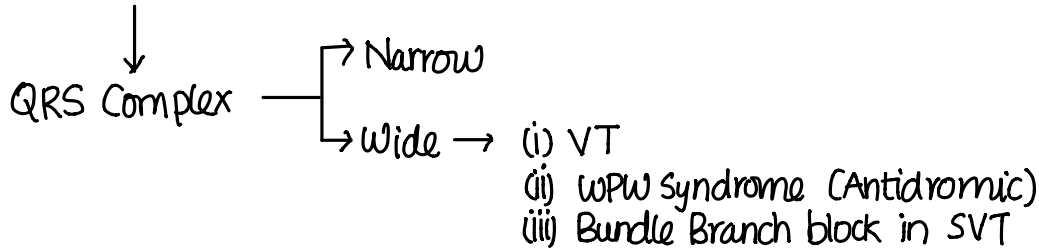
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NARROW QRS COMPLEX TACHYCARDIA

TACHYCARDIA : Rate of ECG >100 /minutes



NARROW QRS COMPLEX TACHYCARDIA

BASED ON ORIGIN

- (i) SINUS TACHYCARDIA
- (ii) FOCAL ATRIAL TACHY.
- (iii) MULTIFOCAL ATRIAL TACHY (MAT)
- (iv) JUNCTIONAL TACHYCARDIA
(Non paroxysmal)

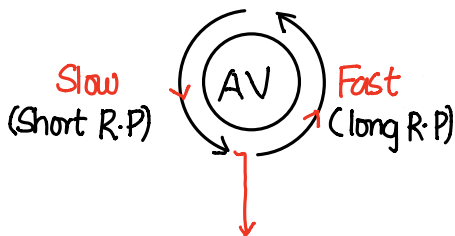
- (v) RE-ENTRY TACHYCARDIA (RT)
 - AV Nodal RT (AVNRT)
 - Atrio Ventricular RT (AVRT)
 - Sinoatrial RT (SART)
 - Interatrial RT (IART)
- (vi) Atrial Flutter
- (vii) Atrial Fibrillation

BASED ON MECHANISM

(i) RE-ENTRY

- Promote in impulse conduction, not in impulse initiation
- 1 single premature atrial impulse that is causing Re-entry

In AVNRT



- Re-entry circuit across AV Node.
- There are two pathways across AV node
 - (i) Slow pathway : short Refractory period.
 - (ii) Fast pathway : Long Refractory period
- Perfectly timed premature atrial complex (trigger) comes to upper part of AV Node

impulse enter into slow pathway ($\because$ short R-P)

Reaches the upper part of Bundle of His

By the time Fast pathway has recovered, $\therefore$ one impulse conducted downwards & other going through fast pathway.

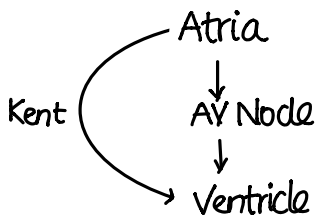
In AVRT

- Always associated with WPW Syndrome

- Perfectly timed premature atrial complex get produced when AV node has recovered & bundle of Kent is Refractory

impulse will travel down to AV Node & activation of ventricle.

At this time Kent get recovered & some impulses traverse back to atrium



✓ In RT, it's the conduction that across AV node is predominantly the issue

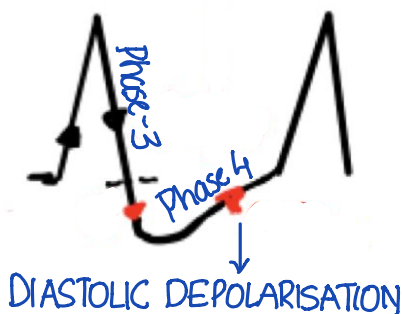
So use **VAGAL MANEUVERS**

AV NODAL BLOCKING AGENT

Electrical DC Cardioversion is also recommended

✓ In structurally normal Heart, RE-ENTRY TACHYCARDIA is **MC**

(ii) ENHANCED AUTOMATICITY



In Pacemaker Cells

✓ Slope of Phase-4 : **Rate of Automaticity**

Enhanced automaticity
or
Enhanced Phase-4
depolarisation

TRIGGER ARRHYTHMIA

Eg: Atrial tachycardia (Focal & Multifocal)
Junctional tachycardia

(i) Problem of ↑ in firing Rate of Automatic cells. No RE-ENTRY circuit.
Problem with INITIATION OF IMPULSE

(ii) Vagal maneuvers
AV node Blockers } No use

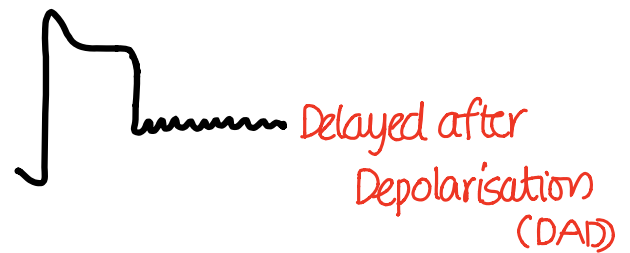
(iii) Electrical cardioversion : No USE

(iv) Most often occurs in structurally abnormal hearts

(iii) TRIGGERED ACTIVITY

- most often due to AFTER DEPOLARISATION

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EAD & DAD can trigger arrhythmia

(i) Triggered activity/EAD : Predominant mechanism of arrhythmia



DUE TO DIGOXIN TOXICITY

(i) Atrial tachycardia ∇ 2:1 block

(ii) Non paroxysmal Junctional tachy- with block.

	Re entry	Enhanced automaticity	Triggered activity
Mechanism	Abnormal pathways in conduction	Abnormality in impulse initiation	Early or late after depolarization
Initiation	Premature impulse	Increase in firing rate of automatic cells	????
Termination	Vagal manoeuvres	No effect	No effect
Underlying condition	Structurally normal heart	Structurally abnormal	Digoxin toxicity
Example	AVNRT / AVRT / SAT / IAT	AT / PAT	Non paroxysmal atrial tachycardia

SUPRA VENTRICULAR TACHYCARDIAS

Narrow QRS Complex ECG
check

Regular RR Interval

- Eg:
- AVNRT
 - AVRT
 - Junctional tachy.
 - Sinus tachycardia
 - Paroxysmal/Focal Atrial tachy
 - Atrial Flutter

Irregular RR interval

- Eg:
- Atrial fibrillation
 - MAT
 - Atrial Tachy ∇ block
 - Atrial Flw ∇ block.

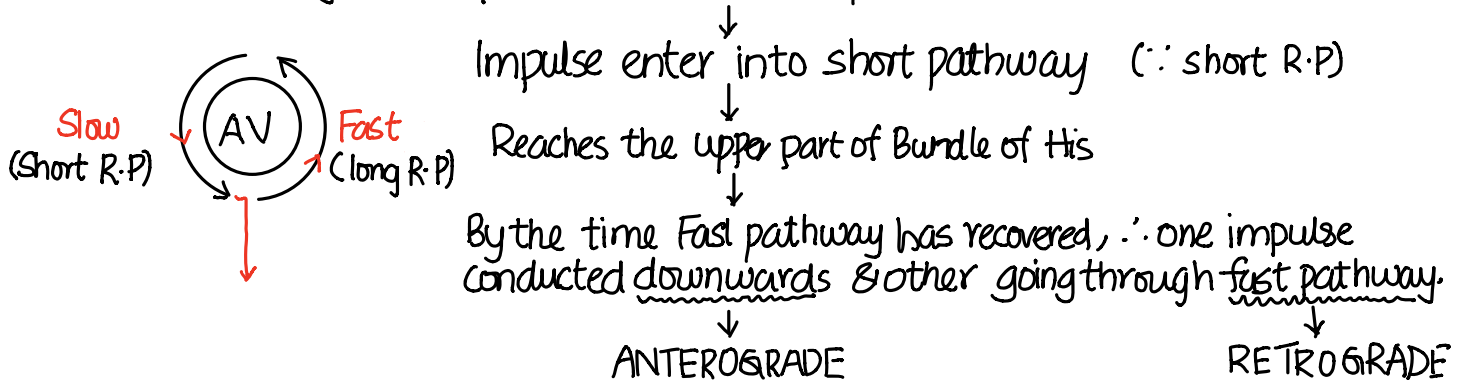
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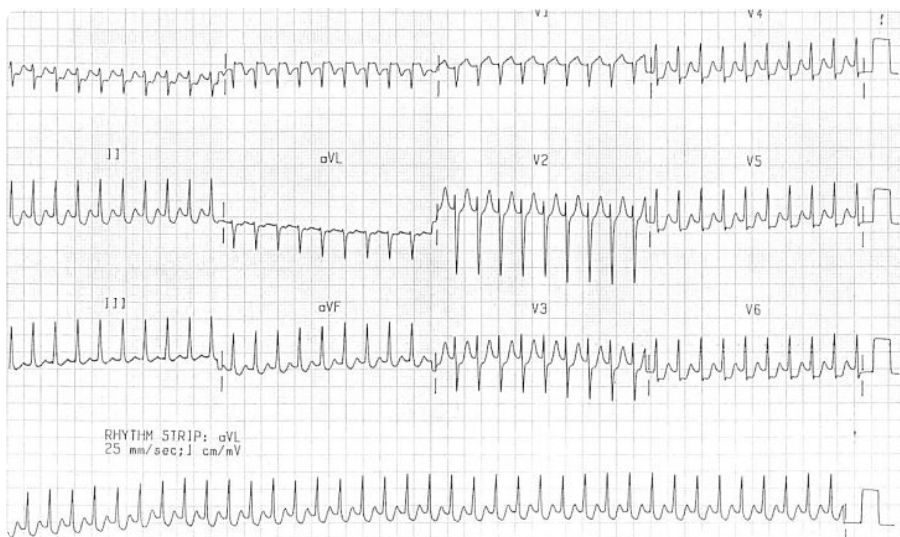
AVNRT - AV Nodal Re-entry Tachycardia

- MOST COMMON TYPE OF SVT DUE TO RE-ENTRY: AVNRT > AVRT > SART > IART
- MOST COMMON TYPE OF SVT: Sinus Tachycardia > Atr. Fibril > AVNRT
- Most often in Structurally normal Heart
- Seen in Females > Males
- Heart Rate : 100-250/minute (MC : 180-250/minute)
- Perfectly timed premature atrial complex Comes to AV Node



- This leads to **SYNCHRONOUS ACTIVATION OF ATRIA & VENTRICLE**

i.e 2/3rd of Patients **P WAVE BURIED INSIDE QRS COMPLEX**



- Narrow QRS Complex
- Rate : $1500/5 = 250-300/\text{min}$
- RR interval = Regular

It's not
Sinus tachycardia $\therefore$ no P wave
Atrial $\therefore$ no P wave
Flutter $\therefore$ no flutter waves

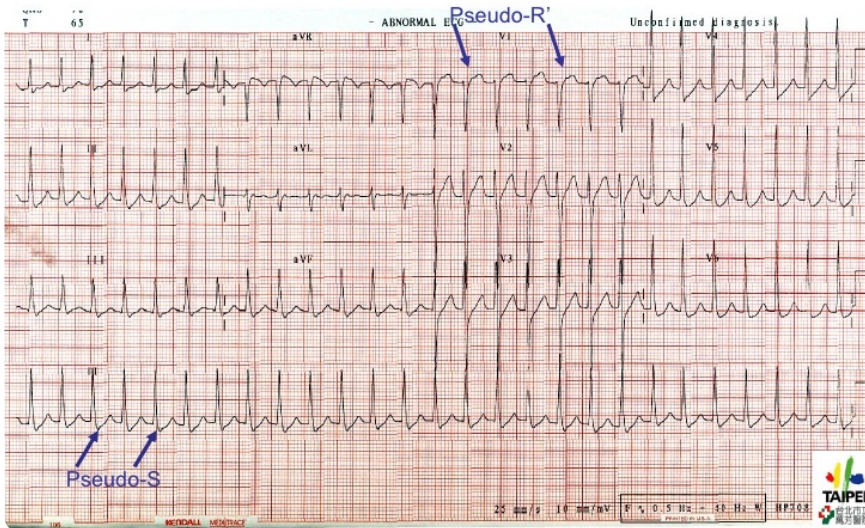
Altered P waves

Junctional : it will be never $> 150/\text{min}$
usually $< 130/\text{min}$

AVRT $\times$: No P wave fall outside

It's
AVNRT $\checkmark$: Correct

S-F AVNRT



Narrow QRS Complex
Regular RR interval
No P waves

∴
AVNRT (or)
AVRT

✓ In AVNRT

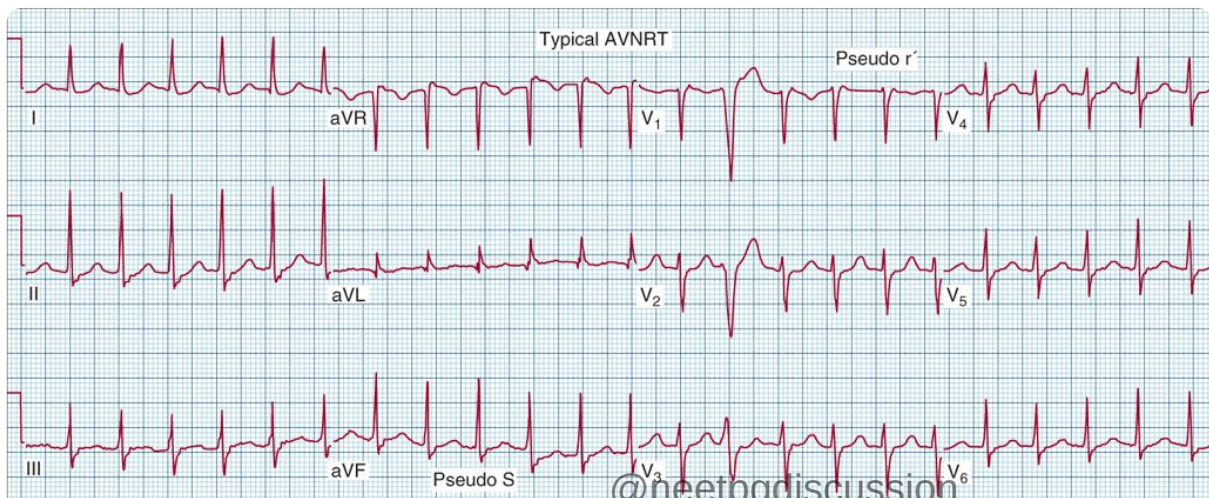
(i) 2/3rd cases : P waves buried inside QRS

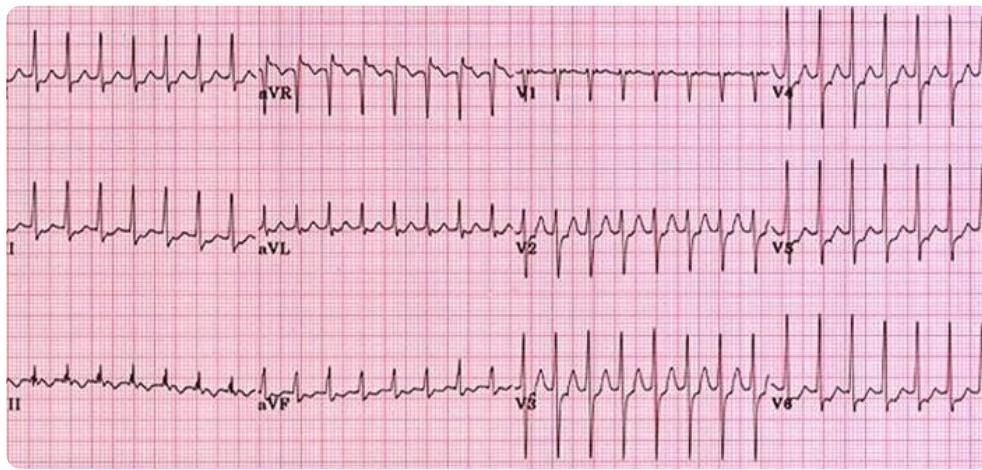
(ii) 1/3rd cases : P waves just outside the QRS complex
(i) MC in the ascending limb of next T wave
called as PSEUDO S-WAVE (MC)

(ii) When it falls on the ascending limb of T wave of V₁ called as PSEUDO R-WAVE

(iii) When it falls before the QRS complex
called as PSEUDO Q-WAVE (Least common)

NOTE : Whenever you see a small deflection
Calculate RP interval = <80ms (<2 small boxes)





Narrow QRS Complex

Regular RR

No pwave

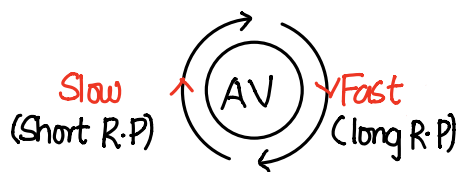
AVNRT/AVRT

TYPICAL AVNRT (Slow-Fast AVNRT) in which

- (i) $\frac{2}{3}$ No P wave
- (ii) $\frac{1}{3}$ Pseudo S, R, Q waves

ATYPICAL AVNRT (Fast-Slow AVNRT)

✓ Here Premature Ventricular complex



Activation of atria by

- (i) RETROGRADE through SLOW pathway
- (ii) ANTEROGRADE through FAST pathway

✓ Here inverted P wave falls before QRS complex

NOTE: Any Narrow QRS complex tachycardia with Regular RR interval Regardless of the Presence or absence of Retrograde P waves is AVNRT.

TREATMENT OF AVNRT, AVRT, AT

✓ **VAGAL MANEUVERS**:

(i) Carotid Sinus Massage

Identifying the carotid artery pulse



Tracing carotid artery pulse to angle of mandible



Gently apply pressure consistently with index & middle finger

(ii) **Valsalva maneuvers**

Pt to exhale against closed glottis & give a fist to abdomen & keep abdomen tensed.

(iii) **DIVING REFLEX**

Dip the head of pt in cold water (vagal stimulation)

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✓ DOC : Adenosine

- Blocks conduction across AV node
- $t_{1/2} = 10 \text{ sec}$
- Opens inward Rectifying K^+ Channels.
- Starting dose : 6mg i/v central vein or if peripheral: **saline chase** i.e. Through one port of canula: 6mg i/v adenosine
Through other port : flush saline as soon as adenosine injected
(do when patient is lying down)

↓
Raise the upper limb

↓
Turn the patient on prone position

- action within seconds
- if not responding try with **12mg i/v**
↓ if not responded
12mg i/v adenosine

- CONTRAINDICATIONS :

(i) COPD or structural Lung disease
as it cause bronchospasm

(ii) Atrial fibrillation $\bar{\wedge}$ WPW Syndrome
blocking of AV node, $\uparrow$ impulse conduction
through aberrant pathway

NOT RESPONDING

Calcium Channel Blocker

(i) **VERAPAMIL** (ii) **DILTIAZEM**

- Preferred as it can be given as bolus dose
- start from 5mg, can give 3-4 times, max 20mg

Disadvantage :

- after giving as bolus dose, give continuous maintenance infusion

Dose : **15-20mg**

NOT RESPONDING

BETA BLOCKERS

METAPROLOL

- 2.5 - 5 mg i/v bolus
- can repeat 3-4 times

NOT RESPONDING

CLASS Ia : **PROCAINAMIDE**

or

CLASS II : **AMIODARONE**

Patient with LV-failure : β -Blocker & CCB C/I

↓
Adenosine not responding

↓
DIGOXIN 0.25-0.5mg

↓ NOT RESPONDING

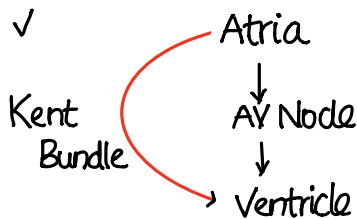
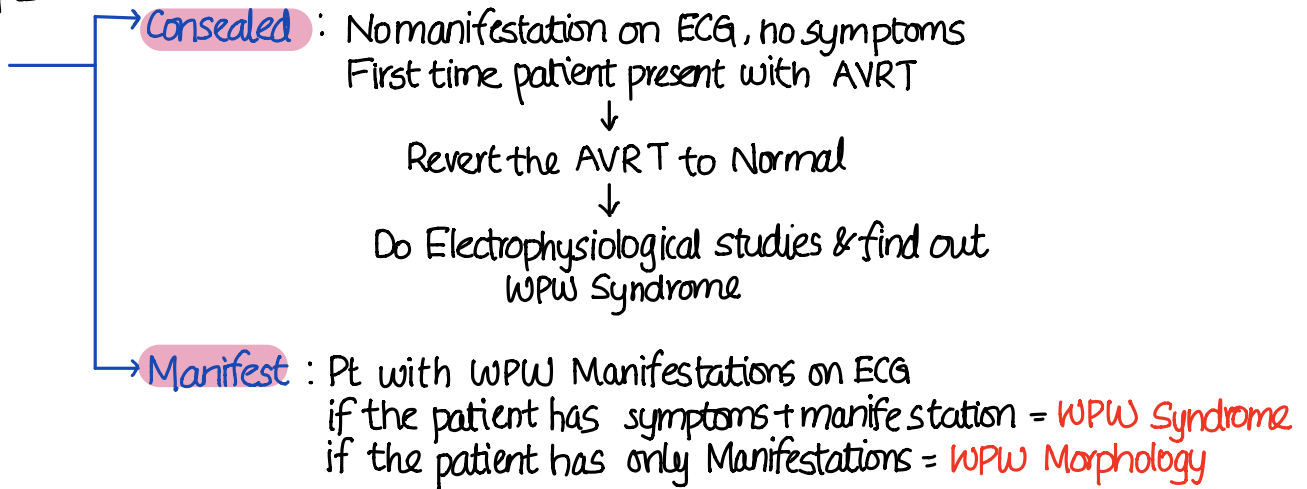
↓
CLASS II : **AMIODARONE**

NOTE : Any arrhythmia $\bar{\wedge}$ Hemodynamic collapse
TOC : Electrical DC Cardioversion (Biphasic)

AVRT : Atrio Ventricular Re-entrant Tachycardia

WPW SYNDROME

✓ 2 types



- Pre-requisite for manifestation of WPW Syndrome
: Impulse transmission anterogradely (from Atria & Ventricle) across the aberrant tract / Bundle of Kent

In CONCEALED WPW : No impulse will travel through Aberrant / Bundle of Kent.

↓ patient

can manifest AVRT at any time

✓ ORTHOGRADE / TYPE A WPW SYNDROME :

- It goes anterogradely via the bypass tract & activate Ventricular myocardium



∴ No AV nodal Delay ⇒ PR interval short

Ventricular myocardial activation is associated with DELTA WAVE via normal myocardial cell not via Purkinji cells

- Some impulse also travel through normal pathway with AV nodal delay



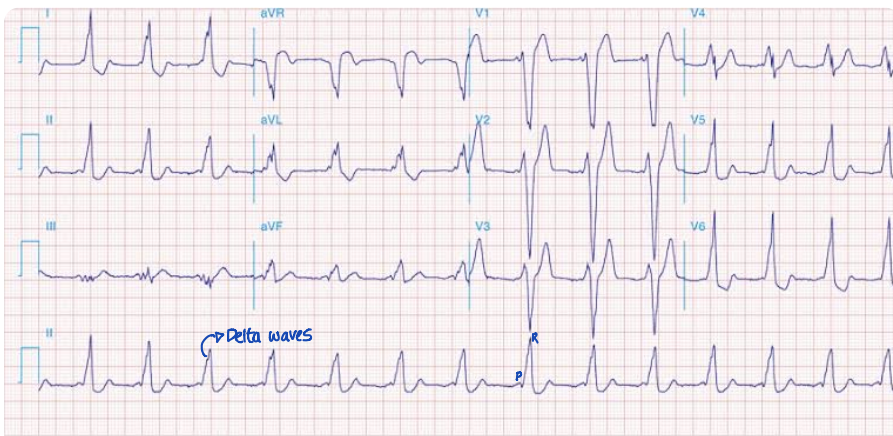
∴ impulse reach the ventricle somewhat later. But this delay at the level of AV node can be compensated by activation of Purkinji System



Catch up the impulse travels along bypass tract



∴ QRS Complex more or less normal with terminal narrow deflection

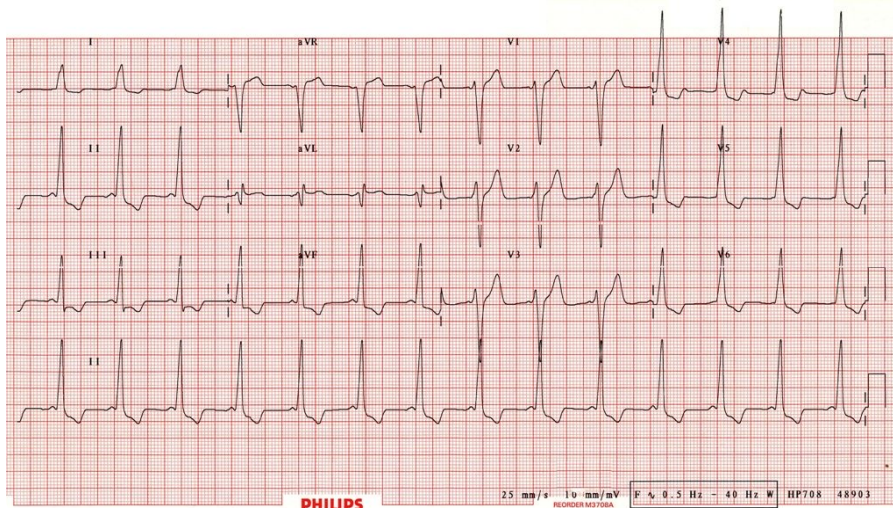


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- Slightly wide QRS Complex
- Short PR interval
- Presence of Delta Waves
 - ↓
 - Upstroke of R wave showing deflection (due to initial Ventricular Depolarisation)
 - or
 - Terminal Narrow deflection of QRS Complex (due to catch up by Purkinji System)

EXAM POINTS ABOUT WPW SYNDROME

- Occurs at any age
- Male > Female
- Normal P wave
- Shortened PR interval
- Bizarre delta waves
- Relatively normal narrow terminal QRS deflection
- Secondary ST & T wave changes



- Near Normal QRS Complex
- Short PR interval
- Terminal Narrow deflection on QRS Complex
- Secondary ST, T changes
- WPW SYNDROME**

LOCATION OF BYPASS TRACT

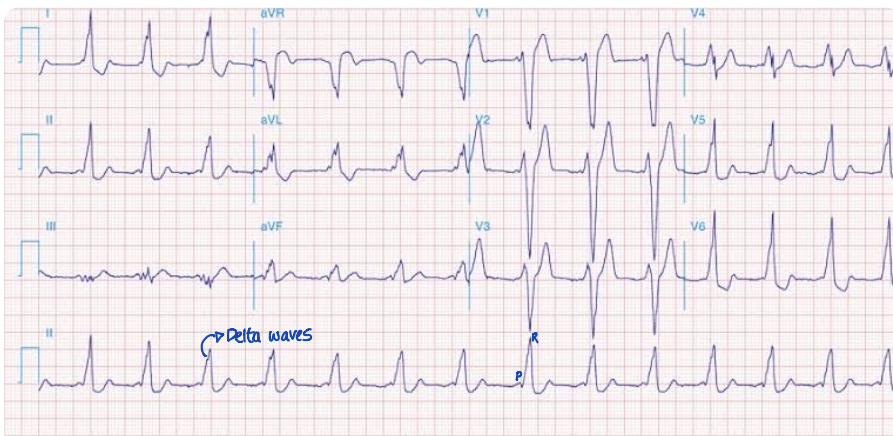
- Now find out the Bypass tract is
 - (i) Lt Sided
 - (ii) Rt sided
- Mostly it's left sided free wall > Lt sided Posteroseptal area > Rt sided

- In Lead V₁: TALL R WAVES (or) PRESENCE OF DELTA WAVES

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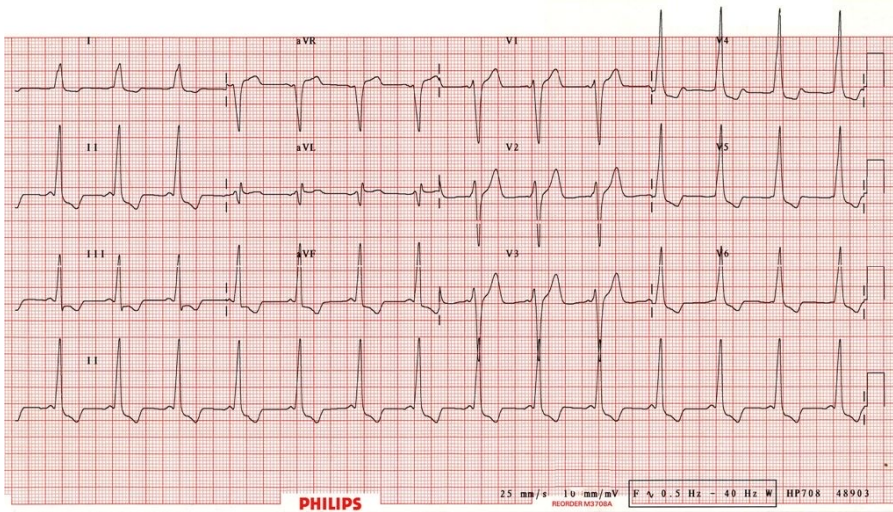
LEFT SIDED PATHWAY / TYPE A WPW

- In Lead V_1 : NEGATIVE WAVES : RIGHT SIDED PATHWAY / TYPE B WPW



Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
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In V_1 :
NEGATIVE WAVES
↓
RIGHT SIDED / TYPE B
WPW



In V_1 :
NEGATIVE WAVES
↓
RIGHT SIDED / TYPE B
WPW

LOCATION OF LEFT SIDED WPW SYNDROME

- either on free wall or Posterobasal area

NEGATIVE or ISOELECTRIC : I, aVL , V_5 or V_6 ⇒ FREEWALL

NEGATIVE or ISOELECTRIC : II, III, aVF ⇒ POSTEROBASAL

✓ Size of DELTA WAVES depend upon amount of myocardium activated.

- Left sided tract has to travel longer distance

↓
Activates less of Myocardium

↓
∴ small Delta wave

- Rt sided tract has to travel lesser distance

↓
Activates more of myocardium

↓
∴ Prominent DELTA WAVES

- Left sided path activates Lt side First

↓
∴ A_2 occurs early. Then P_2 (normal)

↓
 A_2 - P_2 interval widened = WIDE SPLIT OF S_2

- Rt sided path activates Rt side first

↓
∴ P_2 occurs early. A_2 occurs normal

↓
 A_2 - P_2 interval shortened =

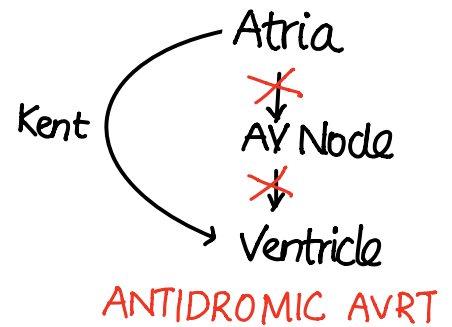
REVERSE SPLIT OF S_2
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ARRHYTHMIAS IN WPW SYNDROME

- ✓ 2 arrhythmias (i) AVRT (ii) Atrial Fibrillation

✓ ANTIDROMIC AVRT

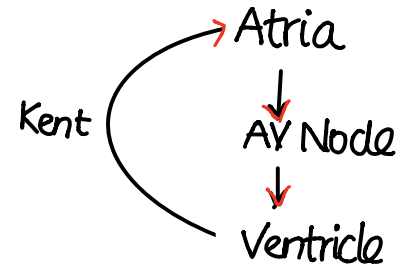
- if the premature atrial complex passes through bypass tract & reach ventricle because the AV Nodal pathway is refractory



↓
We will get **WIDE QRS Complex**
(as it's not conducted through purkinji System)

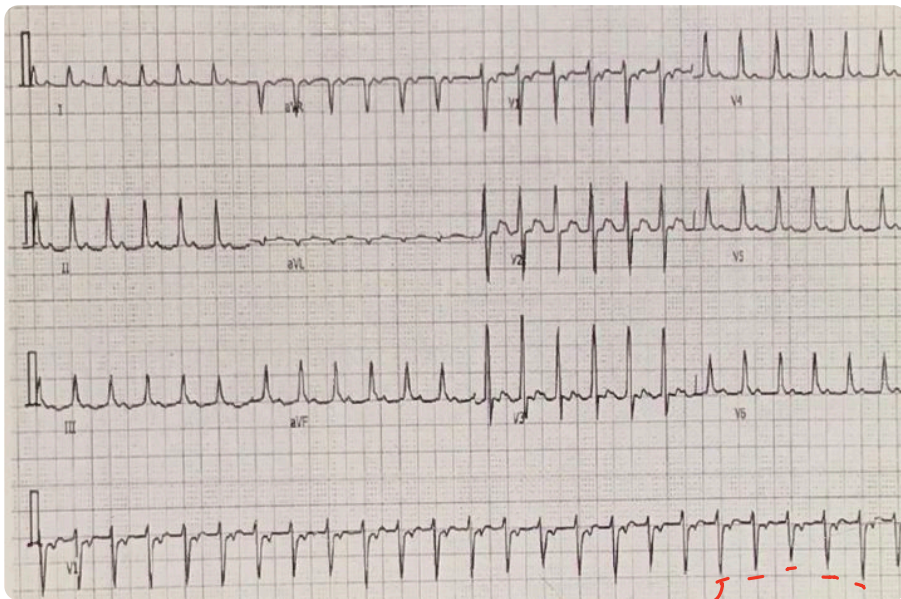
✓ ORTHODROMIC AVRT

- if the impulses are all going via AV node & not going through bypass tract



↓
After reaching Ventricle, these impulse go retrogradely back to atria

- ↓
NARROW QRS Complex (No delta waves)
- Atria & Ventricle will not get activated synchronously
↓
P wave will fall outside the QRS complex



Narrow QRS Complex

Regular RR interval

No Normal Morphology P wave

↓
∴ AVNRT (or) AVRT
P wave falling outside QRS
∴ AVNRT (or) AVRT

In an AVNRT

P wave fall in ascending limb of T (most commonly) such that RP interval is less than <80ms

In an AVRT

Separately seen P wave
RP interval is more than >80ms

Here RP interval >80ms ∴ AVRT

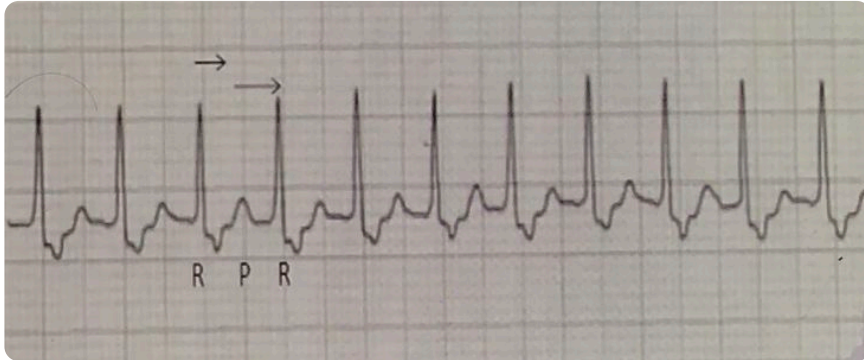
@neetpgdiscussion

NOTE:

- If RP is short: AVNRT v/s AVRT
- ↓ ↓
- RP interval: <80ms >80ms

- In AVRT
RP interval - Short
PR interval - Long

- In Atrial Tachycardia
RP interval: Long
PR interval: Short



- Narrow QRS Complex
- Regular RR interval
- Separate P wave outside QRS
- $PR > RP$
 - ↓
 - ∴ it's not Atrial tachycardia
- RP interval > 80ms
 - ↓
 - ∴ it's AVRT

MANAGEMENT: AVRT

- acute management is same as that of AVNRT
- AVNRT occurs in structurally normal heart, but AVRT Require the patient take him to Electrophysiology & rule out WPW Syndrome
- In orthodromic AVRT : Adenosine
- Antidromic AVRT : Procainamide / Ibutilide (Ia or III)
- Radiofrequency ablation : Definitive treatment
- Recurrent Antidromic AVRT : FLECAINIDE
- WPW Syndrome with Atrial Fibrillation
 - TOC : Electrical Cardioversion
 - DOC : Procainamide (or) Ibutilide
 - C/I : Never give drugs which blocks AV node (No digoxin, BB, CCBs)
 - ↓
 - Because it may precipitate V. Fibrillation

NOTE:

- Pt with concealed WPW syndrome present with ORTHODROMIC AVRT
- Pt with manifest WPW syndrome present with ANTIDROMIC AVRT

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Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
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Narrow QRS Complex
RR intervals are irregular
No morphological P wave

↓
Dx : Atrial Fibrillation
in WPW Syndrome.

AVNRT

- More common
- Structurally normal heart
- No P wave seen
- if P wave seen
RP < 80ms
- Blocks may be seen
- Electrical alternans is not seen

AVRT

- Less common
- Structurally abnormal heart
- P wave seen outside QRS
- P wave seen
RP > 80ms
- No block in AVRT
- Electrical alternans is seen

ATRIAL TACHYCARDIA

Narrow QRS Complex

↓
Regular RR Interval

↓
P wave definite normal morphology
No P wave
Abnormal P wave

→ SINUS TACHYCARDIA

→ AVRT, AVNRT, JUNCTIONAL Tachy

→ Atrial Tachycardia

↓
Rate is much less

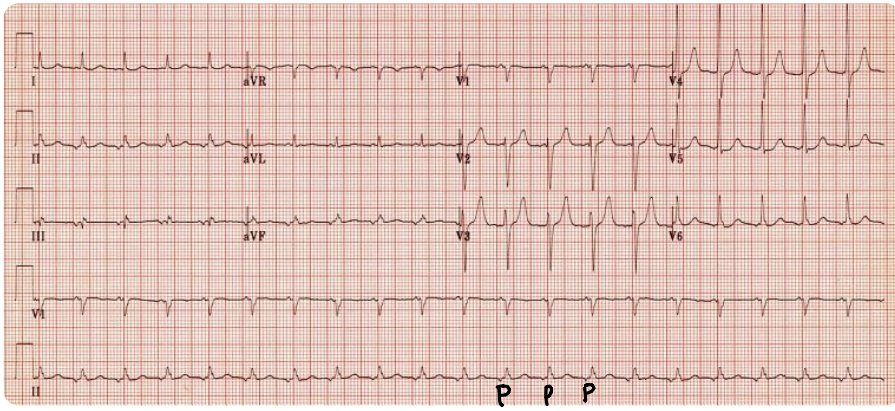
✓

- Focal/Paroxysmal Atrial Tachycardia (PAT) : Regular RR interval
EXCEPTION:
if there is block: Irregular RR interval
- Multifocal Atrial Tachycardia (MAT) : Irregular RR interval

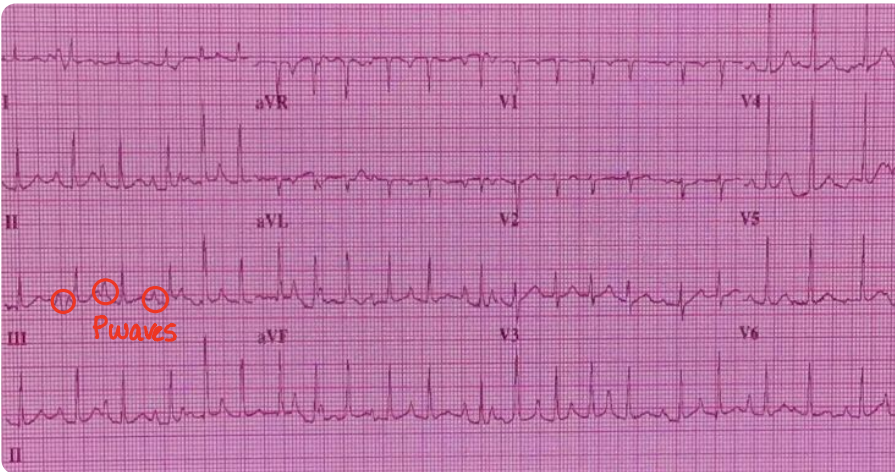
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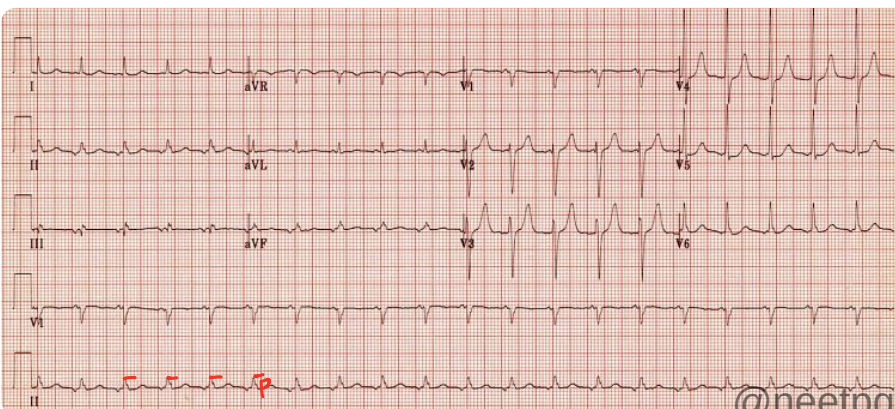
Notespaedia.com
More than just notes...



- Narrow QRS Complex
- Rate = $1500/11 = 130-140$ (generally low)
- Regular RR interval
- Small inverted P wave before the QRS complex (abnormal P wave)
- It's not Junctional: no P wave $RP > PR \therefore$ not AVNRT/AVRT
- $\therefore$ It's focal Atrial Tachycardia.



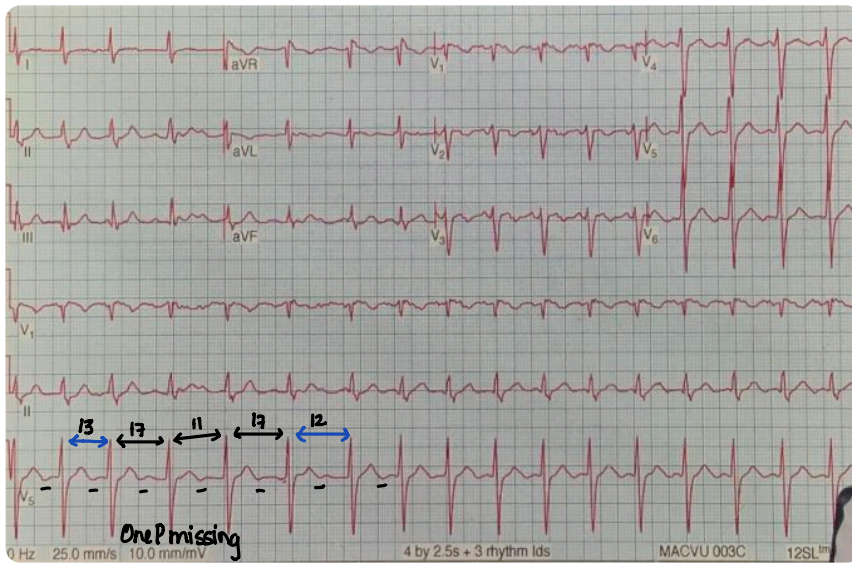
- Narrow QRS Complex
- Irregular QRS complex
- 3 or more different P wave morphology
- $\downarrow$
- Dx: Multifocal Atrial tachycardia



- Narrow QRS complex
- Rate = $1500/12 = 120-130$
- Regular R-R Interval
- Inverted P waves

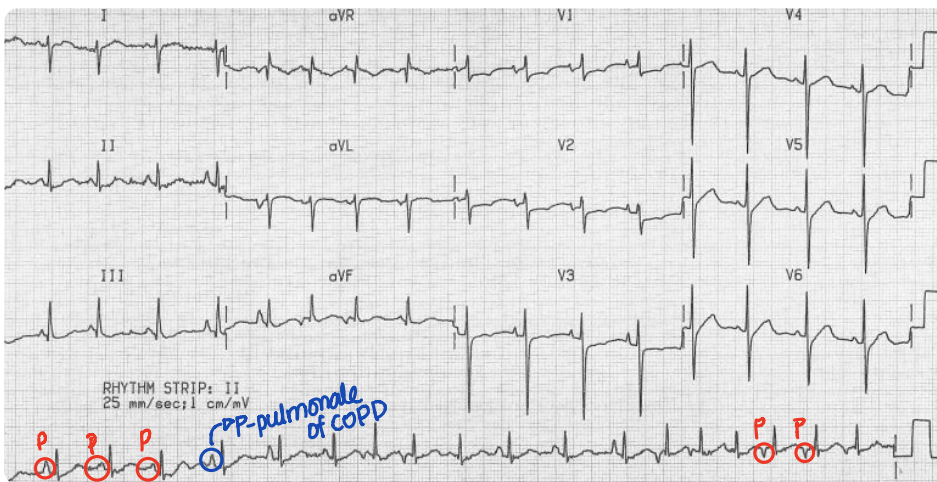
$\downarrow$
Dx: FAT

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- Narrow QRS Complex
- Irregular RR interval
- P waves with same morphology
some P waves are not conducted

Atrial Tachycardia with block

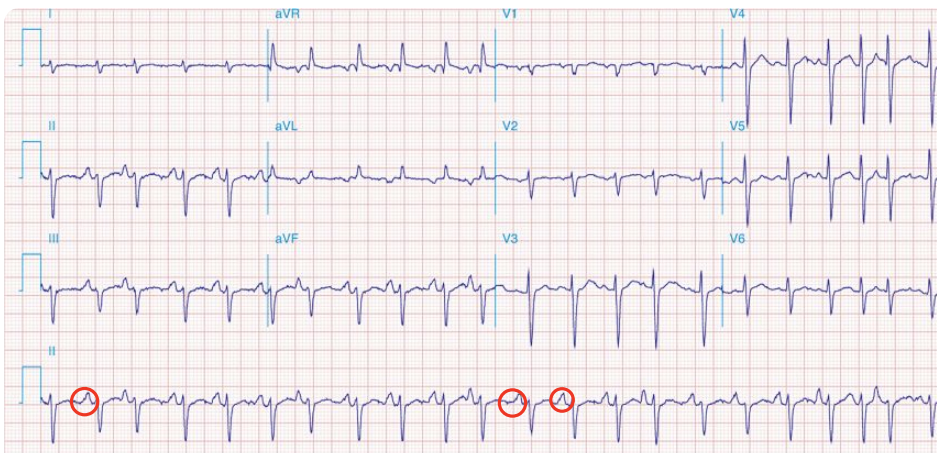


- Narrow QRS complex
- 3 or more different types of P waves

Dx : MAT with additional features of COPD.

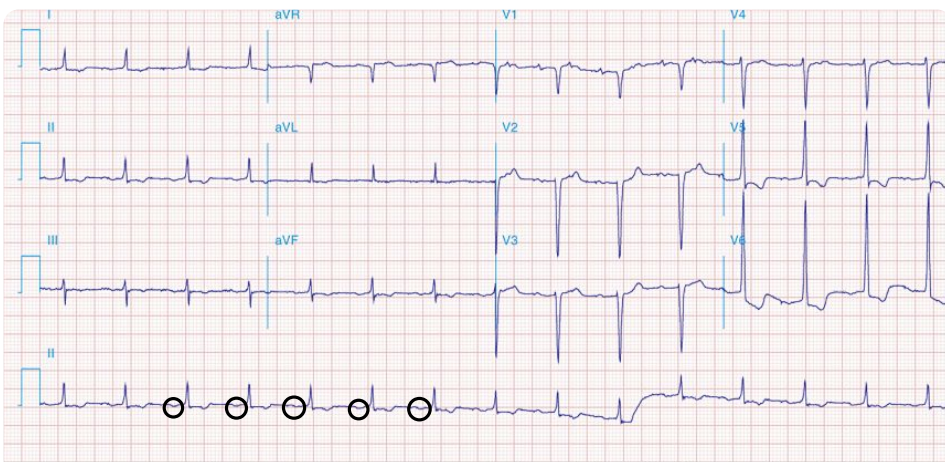
P-Pulmonale P wave:

Tall P waves



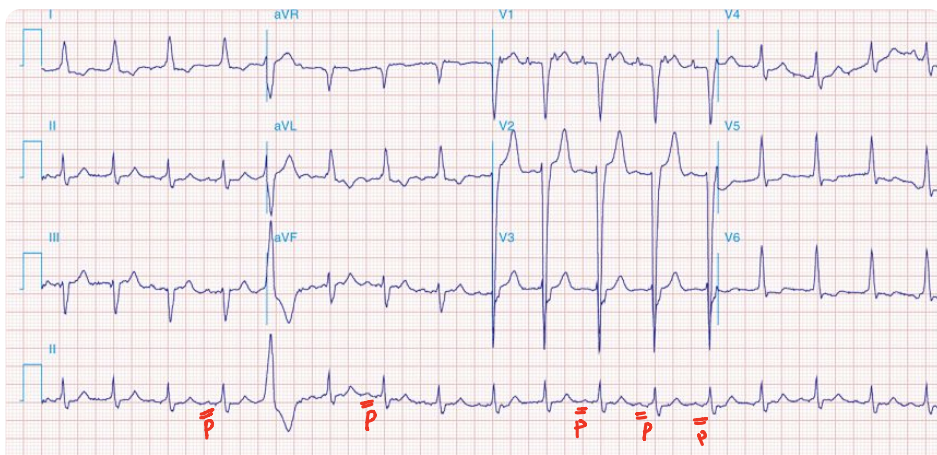
- Inverted QRS complexes
- Irregular RR interval
- 3 or more different P wave morphology

Dx : MAT



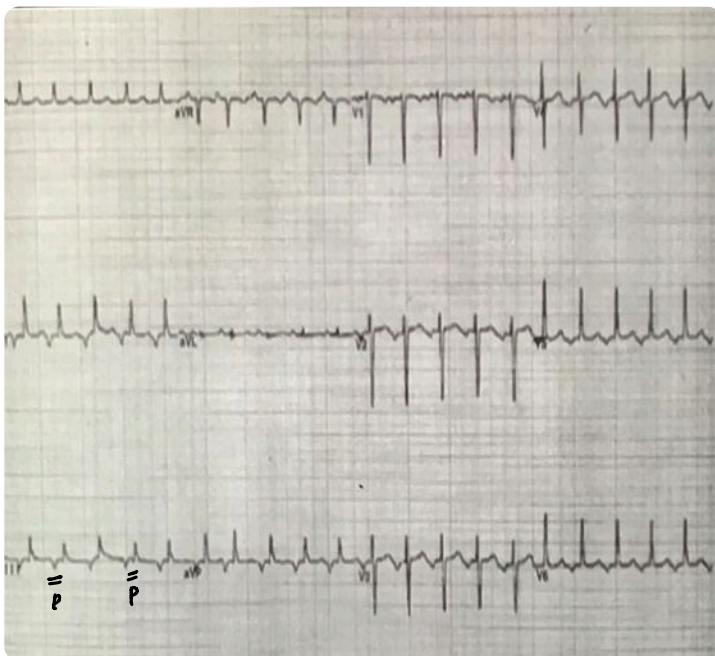
Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
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- NARROW QRS COMPLEX
- IRREGULAR R-R INTERVAL
- SAME MORPHOLOGY P WAVE
- SOME P WAVES ARE NOT CONDUCTED
(2 paves only 1 conducted)
Dx: ATRIAL TACHYCARDIA
WITH 2:1 BLOCK



Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
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- NARROW QRS COMPLEX
 - REGULAR RR INTERVAL
Rate = $1500/15 = 100$
 - ALMOST SAME MORPHOLOGY P WAVE
- ↓
- ATRIAL TACHYCARDIA $\bar{\sim}$ NO BLOCK



- INVERTED P WAVES
- NARROW QRS COMPLEX
- REGULAR R-R INTERVAL

↓

DIAGNOSIS : ATRIAL TACHYCARDIA

@neetpgdiscussion

- Mechanism behind Atrial Tachycardia : ENHANCED AUTOMATICITY

- ∴ AV NODAL BLOCKING AGENTS
- VAGAL MANEUVERS
- DC CARDIOVERSION

} will not Respond

- Here some kind of structural abnormality are present

MOST OFTEN OCCURS IN: MI

OBSTRUCTIVE LUNG DISEASE: (MC: MAT)

ATRIAL TACHYCARDIA with 2:1 AV BLOCK

DIGOXIN

When nebulising patient of COPD with HR >120 /minutes leads to arrhythmia, Atrial tachycardia (MC:MAT)

MECHANISM: TRIGGERED ACTIVITY

- Abnormal & discrete P waves
- Pathway is independent of AV CONDUCTION
- RATE is < 200

- DEFINITIVE TREATMENT: Catheter Ablation

ECG FINDINGS & TREATMENT OF PAT

1. Regular Narrow QRS complex tachycardia
2. P WAVES : UNIFORM IN CONFIGURATION BUT DIFFERENT IN MORPHOLOGY
3. **ISOELECTRIC BASELINE BETWEEN 2P WAVES**
4. PR INTERVAL : Prolonged
VARYING Degrees of AV BLOCK Present

NOTE :

IRREGULAR
RR

- MAT
- PAT 5 BLOCK

- ADENOSINE

- will not respond to Atrial tachycardia.
But still we use because Rate will come down
- But generally work in AVNRT, AVRT but not in AT.

- TREAT UNDERLYING PROBLEM (Structural Heart disease, COPD, Digoxin etc)
- REVERT THIS WITH CLASS $I_A/I_c/III$: PROCAINIDE/FLECAINIDE/IBUTILIDE/AMIODARONE
DOC : IBUTILIDE 1mg i/v for 10 minutes
with H.F : AMIODARONE

ECG FINDINGS & TREATMENT OF MAT

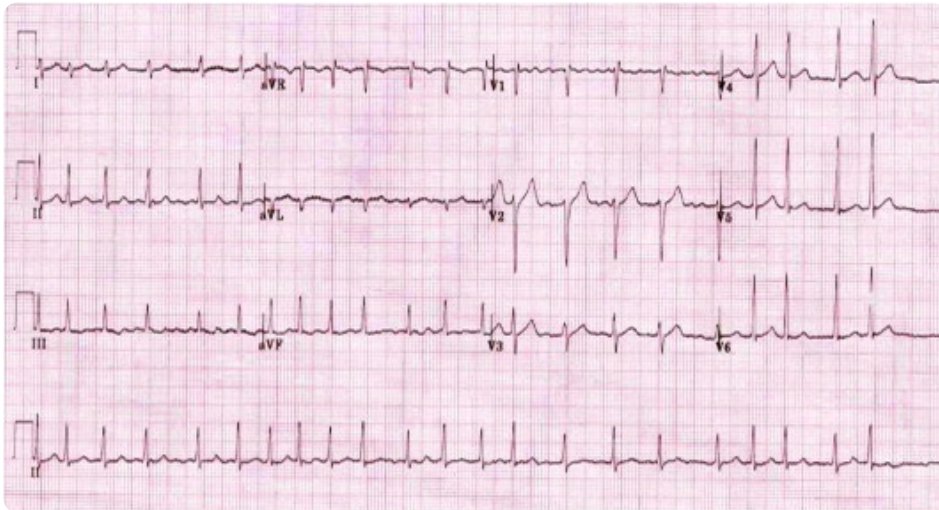
- 1. 3 or more different morphology Pwaves
- 2. ISOELECTRIC BASE LINE
- 3. COPD / Theophylline
- TREAT UNDERLYING PROBLEM
- NO ROLE of ELECTRICAL CARIOVERSION @ne
- CALCIUM CHANNEL BLOCKERS/ BETABLOCKERS USED

MAT V/S AF

* does not require ANTICOAGULANT
* Require ANTICOAGULANT

3 or more different P wave morphology

ATRIAL FIBRILLATION



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVALS
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

ATRIAL FIBRILLATION

- 3RD MC SVT
- MC RE-ENTRY ARRHYTHMIA

- HALL MARK: IRREGULARLY IRREGULAR Ventricular Rhythm
Ventricular Rate : 400-450/min
Atrial Rate : 100-160/min

CLASSIFICATION

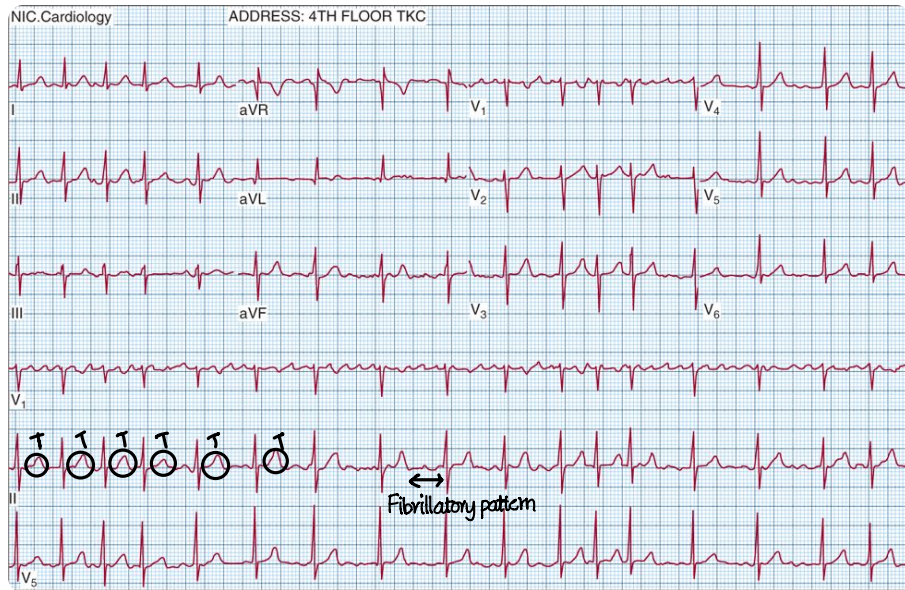
Terminology	Clinical features	
Initial event (first detected episode)	Symptomatic Asymptomatic Onset unknown	Rhythm/Rate
Paroxysmal	Spontaneous termination <7 days and most often <48 hours	Rhythm Control
Persistent	Not self-terminating Lasting >7 days or prior cardioversion	Rhythm or Rate control
Permanent ('accepted')	Not terminated or >1yr Terminated but relapsed No cardioversion attempt	Rate Control

LONE AF :

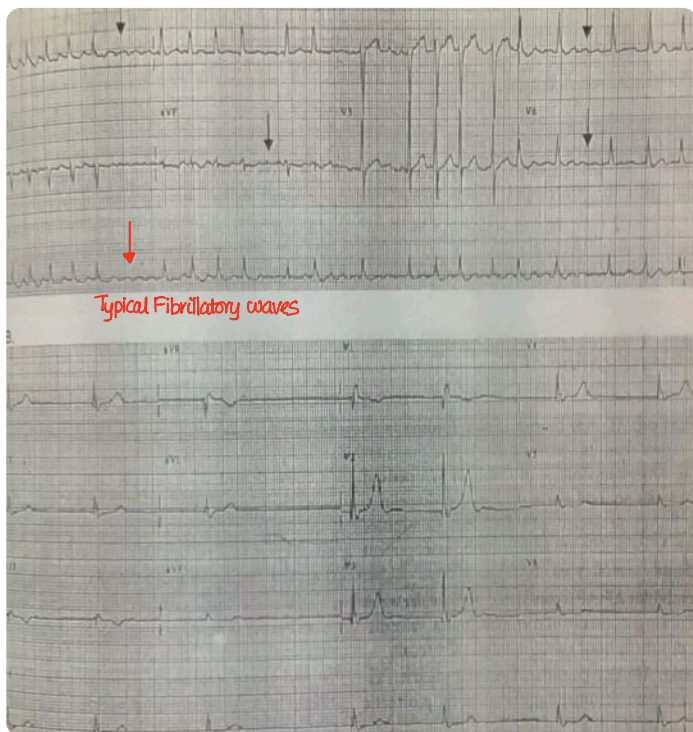
- AF which occurs <60yrs
- No structural heart disease
- No hemodynamic instability
- No co-morbidities

CAUSES OF ATRIAL FIBRILLATION

- MC Valvular heart disease causing AF: MR > MS
 - BOTH MS & MR associated with AF
 - ↓
 - MORE STRONGLY ASSOCIATED WITH AF
 - MR incidence is > MS
 - ∴ MC Valvular disease causing AF MR
- MC ACUTE CONDITION WHICH LEAD TO AF: PNEUMONIA (LRTI)
ALCOHOL INTAKE
THYROTOXICOSIS
- MC CONGENITAL HEART DISEASE ASSOCIATED WITH AF: ASD (Atrial Septal Defect)
- MC ARRHYTHMIA SEEN AFTER POST OPERATIVE SURGERY: ATRIAL FLUTTER > FIBRI...



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES
 - ↓
 - ATRIAL FIBRILLATION



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES
 - ↓
 - ATRIAL FIBRILLATION

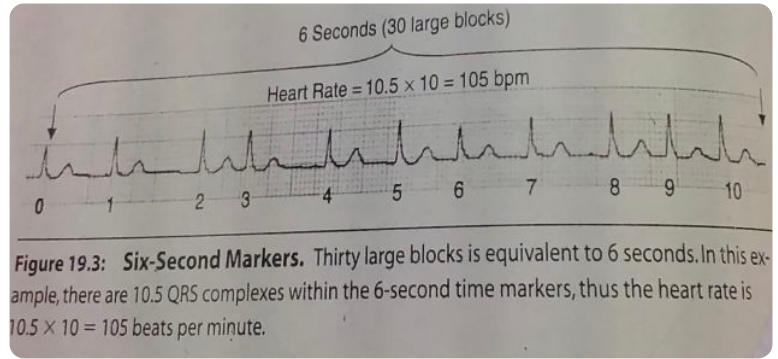
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HR IN ATRIAL FIBRILLATION

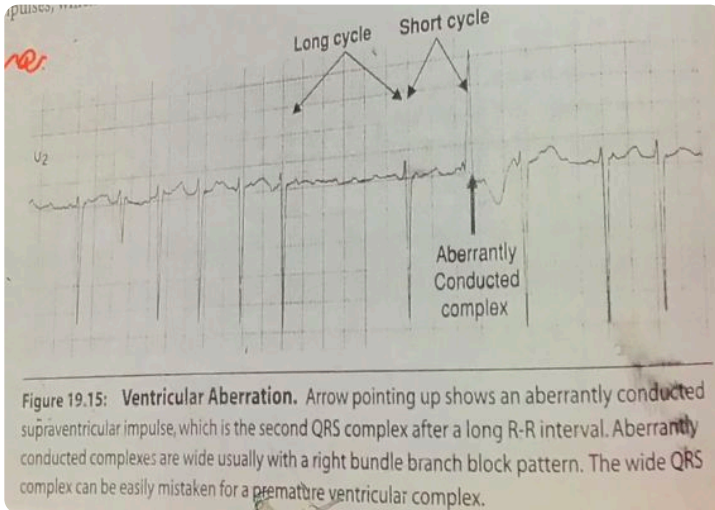
6-second Rule:

$$HR = N \times 10$$

N = No. of QRS Complexes in 30 large boxes or 6 seconds



ASHMANN PHENOMENON



In AF after a LONG CYCLE when you are having a SHORT CYCLE, next IMPULSE IS FINDING VENTRICLE REFRACTORY (because it's not recovered from previous R-P)

NEXT IMPULSE WILL BE CONDUCTED DOWN THE VENTRICLE Called as **ABERANCY**

This Phenomenon is known as **ASHMANN PHENOMENON**

CHADS₂-VAS SCORE

- ✓ ONE OF THE MOST COMMONEST CAUSE OF EMBOLIC STROKE IS AF
- ✓ CHADS₂ VAS SCORE Predict the RISK OF EMBOLIC STROKE after AF

Risk Factors	Points	CHA ₂ DS ₂ -VASc Score	Estimated Annual Stroke Rate ^a
C - congestive heart failure	1	0	0
H - hypertension	1	1	1.3%
A - age ≥75 y	2	2	2.2%
D - diabetes mellitus	1	3	3.2%
S - stroke or TIA, embolus	2	4	4.0%
V - vascular disease	1	5	6.7%
A - age 65-75 y	1	6-9	>9%
Sex - female	1		

Anticoagulants	Mechanism	Excretion	Dosing Considerations	Risk/Benefit
Warfarin	Vitamin K antagonist	Liver	Adjusted to INR 2-3 Days to therapeutic effect Multiple drug/food interactions (e.g., amlodarone)	Major hemorrhage: 1% per year Intracranial hemorrhage: 0.1-0.6% per year Risk of bleeding increases with INR >3.5 Inexpensive
Dabigatran ^b	Thrombin inhibitor	Kidney CCr >30 mL/min CCr 15-30 mL/min	150 mg bid 75 mg bid P-glycoprotein substrate (Inducers - rifampin, reduce concentration) (Inhibitors - amlodarone, verapamil, dronedarone, quinidine) Proton pump inhibitors may reduce absorption	Onset of action within hours No reversal agent for bleeding
Rivaroxaban	Xa inhibitor	Kidney CCr ≥50 mL/min CCr 15-50 mL/min	20 mg daily 15 mg daily	No reversal agent for bleeding
Apixaban	Xa inhibitor	Kidney and liver Cr >1.5 mg/dL	P-glycoprotein substrate 2.5 mg bid	No reversal agent for bleeding

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✓ As CHAD₂ VAS Score increases it increases risk of Stroke

✓ SCORE ZERO : NO ANTICOAGULANT
SCORE ONE : ASPIRIN Alone
SCORE >2 : ORAL ANTICOAGULANT

✓ Pt with score >2

(i) WARFARIN

(ii) DIRECT THROMBIN INHIBITOR : DABIGATRAN -MOST PREFERRED

(iii) ORAL X_A INHIBITOR : RIVAROXABAN
APIXABAN

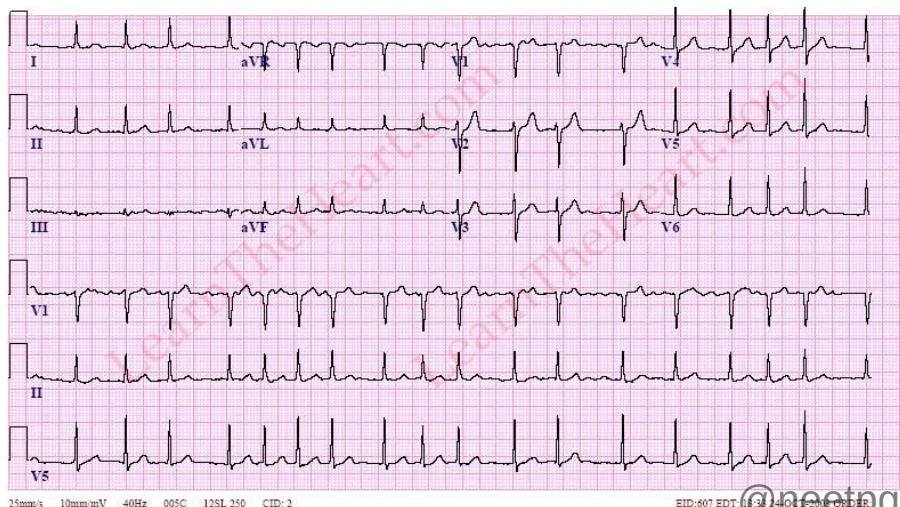
✓ End-stage CKD not on dialysis : ONLY DRUG WARFARIN

HAS-BLED SCORE

- It determine the BLEEDING RISK after Anticoagulant Therapy

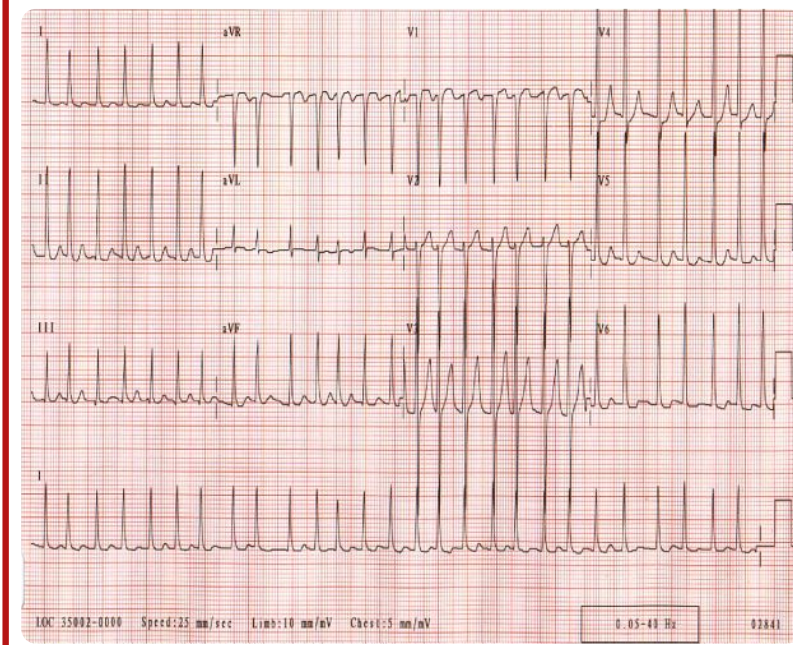
Bleeding Risk Assessment (HAS-BLED Score)	
	Points
• Hypertension (> 160 mm Hg systolic)	1
• Abnormal renal or hepatic function	1-2
• Stroke	1
• Bleeding history or anemia	1
• Labile INR (TTR < 60%)	1
• Elderly (age > 75 years)	1
• Drugs (antiplatelet, NSAID) or alcohol	1-2
• High risk (> 4%/year) >4	
• Moderate risk (2-4%/year) 2-3	
• Low risk (< 2%/year) 0-1	

Dose Selection of Oral Anticoagulant Options for Patients with Nonvalvular AF and CKD (Based on Prescribing Information for the United States)*				
Renal Function	Warfarin	Dabigatran†	Rivaroxaban†	Apixaban†
Normal/Mild Impairment	Dose adjusted for INR 2.0-3.0	150 mg BID (CrCl >30 mL/min)	20 mg QD with the evening meal (CrCl >50 mL/min)	5.0 or 2.5 mg BID‡
Moderate Impairment	Dose adjusted for INR 2.0-3.0	150 mg BID or 75 mg BID§ (CrCl >30 mL/min)	15 mg QD with the evening meal (CrCl 30-50 mL/min)	5.0 or 2.5 mg BID‡
Severe Impairment	Dose adjusted for INR 2.0-3.0¶	75 mg BID§ (CrCl 15-30 mL/min)	15 mg QD with the evening meal (CrCl 15-30 mL/min)	No recommendation†
End-Stage CKD Not on Dialysis	Dose adjusted for INR 2.0-3.0¶	Not recommended† (CrCl <15 mL/min)	Not recommended† (CrCl <15 mL/min)	No recommendation†
End-Stage CKD on Dialysis	Dose adjusted for INR 2.0-3.0¶	Not recommended† (CrCl <15 mL/min)	Not recommended† (CrCl <15 mL/min)	5.0 or 2.5 mg BID¶



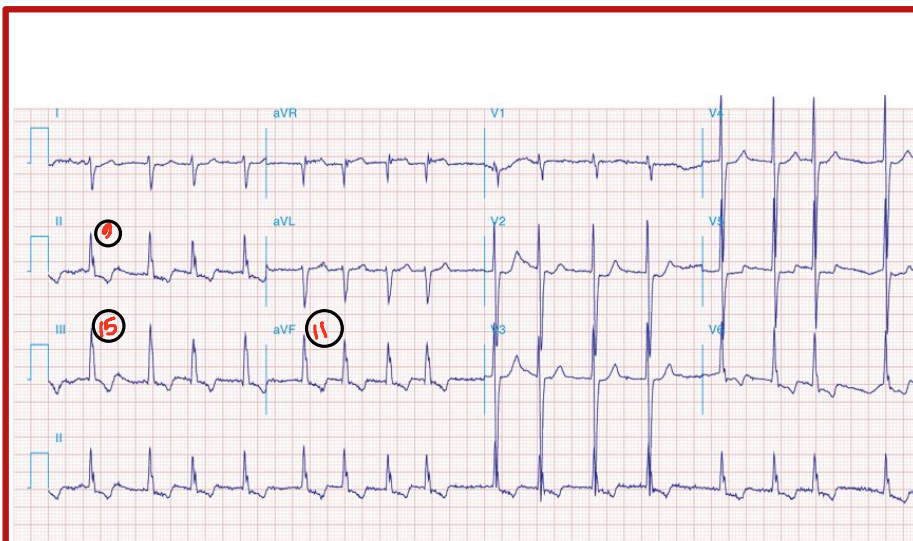
- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓
ATRIAL FIBRILLATION



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓
ATRIAL FIBRILLATION



Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
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- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓
ATRIAL FIBRILLATION

AXIS OF ECG
B/W LEAD III & AVF
↓ ↓
90° - 120°
RAD

MANAGEMENT OF AF

- Rate control & Rhythm control done. Both are having equal efficacy.

- RHYTHM CONTROL FOR

1. YOUNG PATIENT with NEW ONSET AF
2. REVERSIBLE CAUSE FOR AF
3. CHF precipitated by AF
4. LONE AF
5. PATIENT CAN'T TAKE ANTICOAGULANT

- RHYTHM CONTROL

(A) PATIENT HEAMODYNAMICALLY STABLE

Class I_A : PROCAINIDE

Class I_c : FLECAINIDE

Class III : DOBUTILIDE, IBUTILIDE, AMIODARONE

↳ Preferred

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} APPROVED IN PERSISTENT AF

1mg i/v for 10 min.



Notespaedia.com
More than just notes...

PATIENT WITH CAD/CHF/CCF : AMIODARONE or DOFETILIDE or SOTALOL

(B) PATIENT WITH HEAMODYNAMICALLY UNSTABLE

- TOC : DC Cardioversion : BIPHASIC 200J

↓
if it's planned then
give ANTICOAGULANT 3 weeks before

↓
Then do DC Cardioversion

↓
Then give ANTICOAGULANT 4 weeks

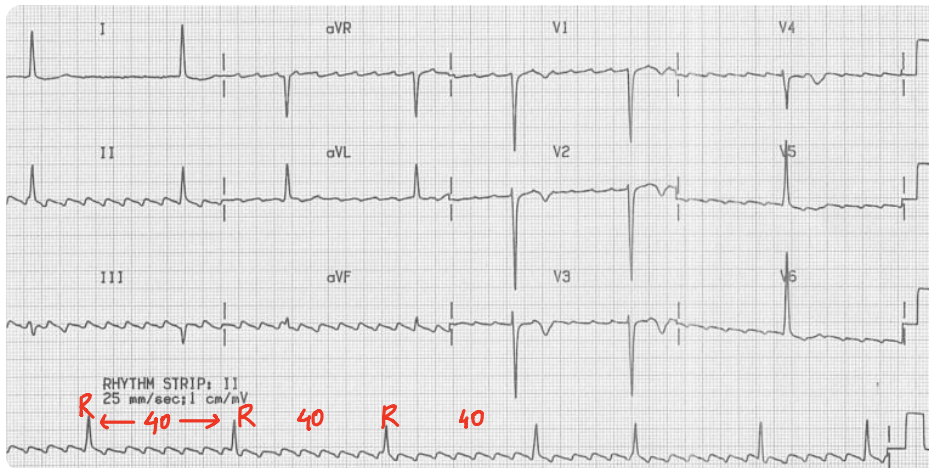
- if PATIENT PRESENTED AFTER 48 HRS

↓
DO TRANSESOPHAGEAL ECHO (TEE) to pick
up the THROMBUS FORMED

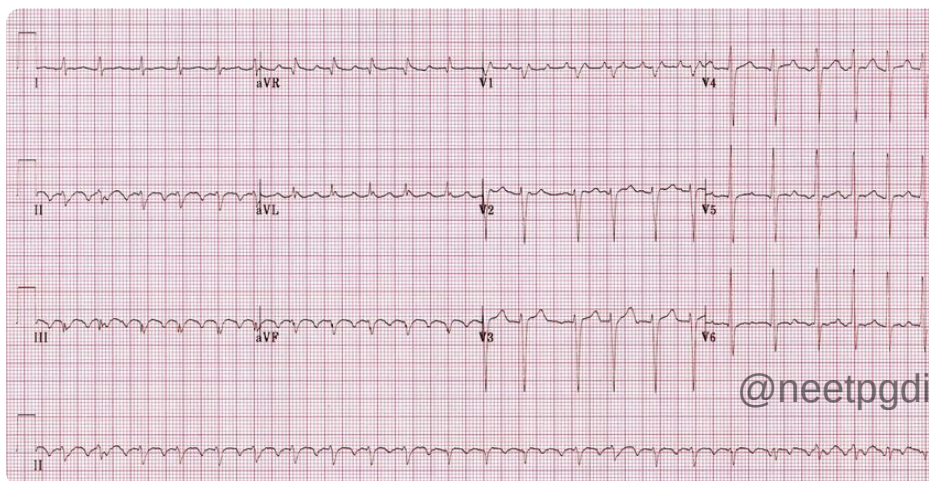
- CATHETER ABLATION :
DEFINITIVE TREATMENT
- MAZE PROCEDURE :
SURGICAL PROCEDURE
- Opening of PULMONARY VEIN is
the site which precipitate Lt
ATRIUM

- RATE CONTROL : CALCIUM CHANNEL BLOCKERS : VERAPAMIL 5mg i/v
BETA BLOCKERS : METOPROLOL (2.5-5 mg i/v)
DIGOXIN
AMIODARONE (can also be used)

ATRIAL FLUTTER



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- FOR 7 P WAVE one R WAVE
↓
inverted
↓
HIGH DEGREE BLOCK
- IRREGULAR BASE LINE
- PP interval : 300-350 ms
- UNDULATING BASE LINE + SAW TOOTHED WAVES = ATRIAL FLUTTER with AV Block

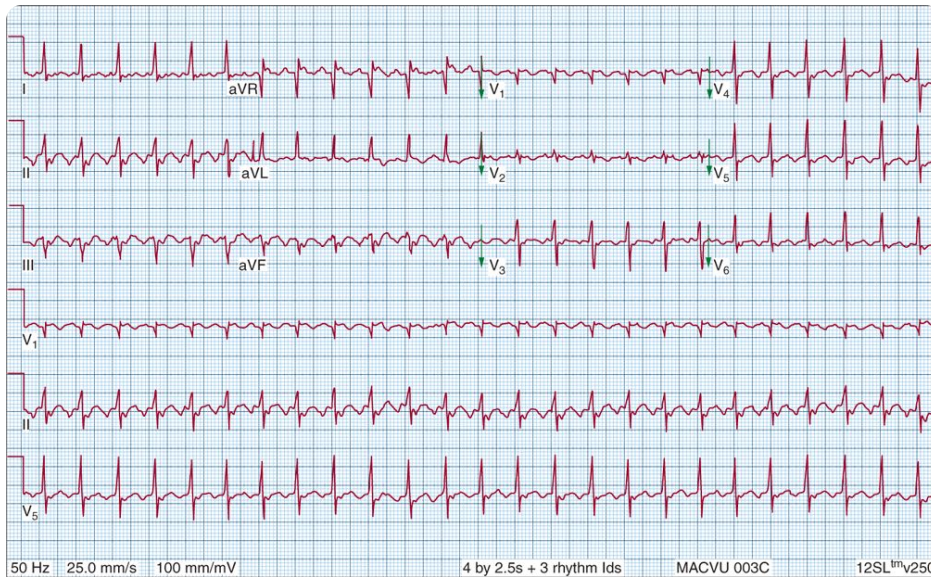


- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- UNDULATING BASE LINE
- SAW TOOTHED/FLUTTER WAVE
- INVERTED P WAVE
INVERTED in LEAD II & type in V1
- ATRIAL FLUTTER with Block

GENERALLY FLUTTER P waves are

POSITIVE IN : V_1
INVERTED IN : LEAD II

} $\therefore$ they are Right sided Flutter & come out through the right wall & go back via Interatrial septum



- NARROW QRS COMPLEX
- REGULAR RR INTERVAL
- Rate : 150/min
- P waves before QRS Complex with ABNORMAL P WAVE
- UNDULATING BASELINE + POSITIVE PWAVE in V_1 + INVERTED P WAVE in II
- ATRIAL FLUTTER with 2:1 block (as the Atrial rate $>300/\text{min}$ it's not Atrial tachycardia)

- SVT Arrhythmia with atrial rate of 250-350/min
- 00% across LATERAL WALL OF RIGHT ATRIUM $\therefore$ P waves are positive in V_1
P waves are negative in II, III, aVF
- VERY REGULAR UNIFORM FLUTTER WAVES with SAW TOOTHED WAVES
- MC ARRHYTHMIA AFTER OPEN HEART SURGERY : Atrial Flutter (in 1 week)
- ATRIAL COMPLEXES OF : CONSTANT MORPHOLOGY AND CYCLE LENGTH
+
UNDULATING BASELINE

MANAGEMENT

- Risk for Stroke present
- $\therefore$ patient with Post 48hrs with ATRIAL FLUTTER
↓
ANTICOAGULATE THE PATIENT
- AV Nodal Blockers : β -Blockers, CCB
↓
↓ impulse coming down to ventricle & make the FLUTTER more PROMINENT
- TOC : DC CARDIOVERSION 25-50J
- IBUTILIDE : MOST EFFECTIVE DRUG IN CONVERTING ATRIAL FIBRILLATION TO NORMAL SINUS RHYTHM
- SITE OF FOCUS : CAVO TRICUSPID ISTHMUS

NOTE :

- For atrial Flutter the Atrial rate $>300/\text{min}$ But for AT it's $<300/\text{min}$
- No role for carotid sinus massage as the Mechanism is not RE-ENTRY

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Notespaedia.com
More than just notes...

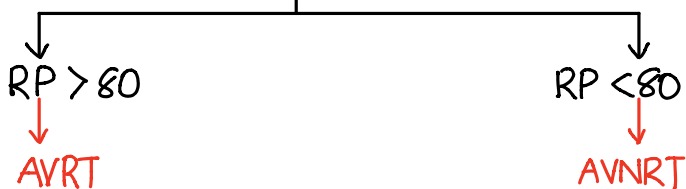
NARROW QRS COMPLEX

REGULAR RR INTERVAL

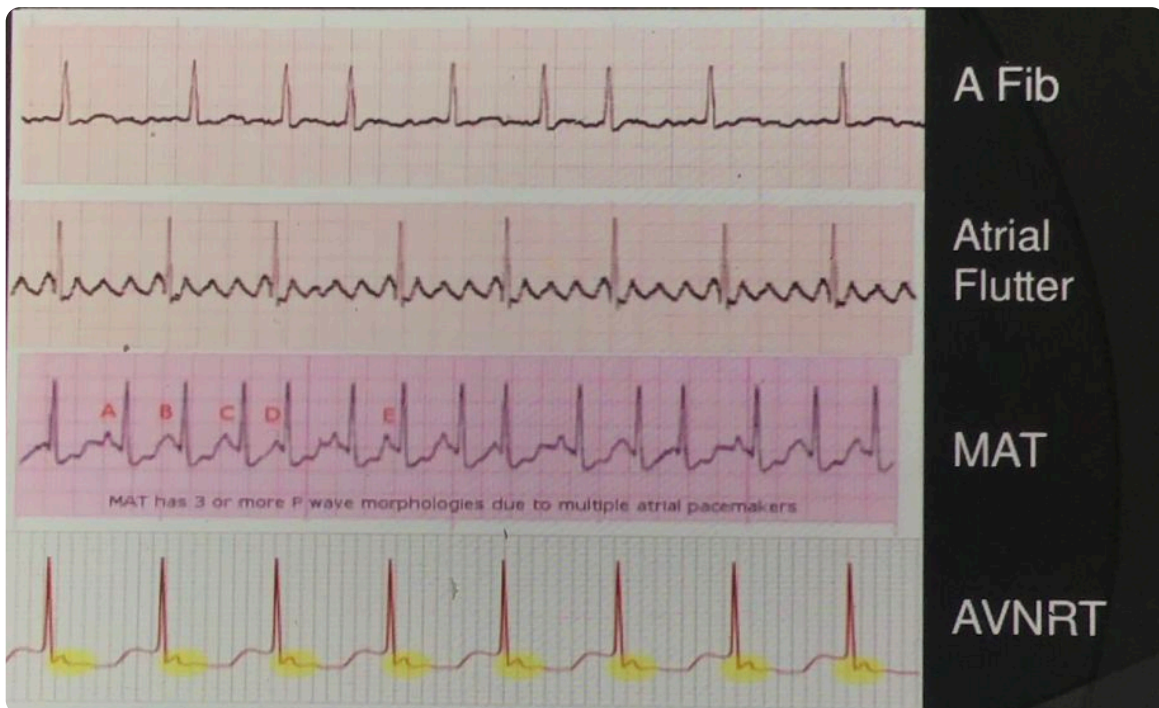
IRREGULAR RR INTERVAL

1. Normal Morphology P wave : **ST**
2. Abnormal Morphology P wave : **AT**
3. No P Wave : **AVNRT / Junctional**
4. P wave outside QRS Complex

1. NO P WAVE : **AF**
2. 3 or More different morphology } **MAT**
P waves
3. FLUTTER } with VARIABLE BLOCK
4. AT



5. UNDULATING BASELINE : **AF**



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Message @drnash1 for group discount upto 50% on any lecture

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WIDE QRS COMPLEX TACHYCARDIA

- ✓ Most commonly it's **VENTRICULAR TACHYCARDIA (VT)**
- ✓ **SUPRAVENTRICULAR IMPULSE** with **BUNDLE BRANCH BLOCK**
- ✓ **ANTIDROMIC AVRT**

VENTRICULAR TACHYCARDIA - MONOMORPHIC

- In Ventricular tachycardia, there is **AV Dissociation** i.e atria & ventricle get separated completely. (**AV ASYNCHRONY**)

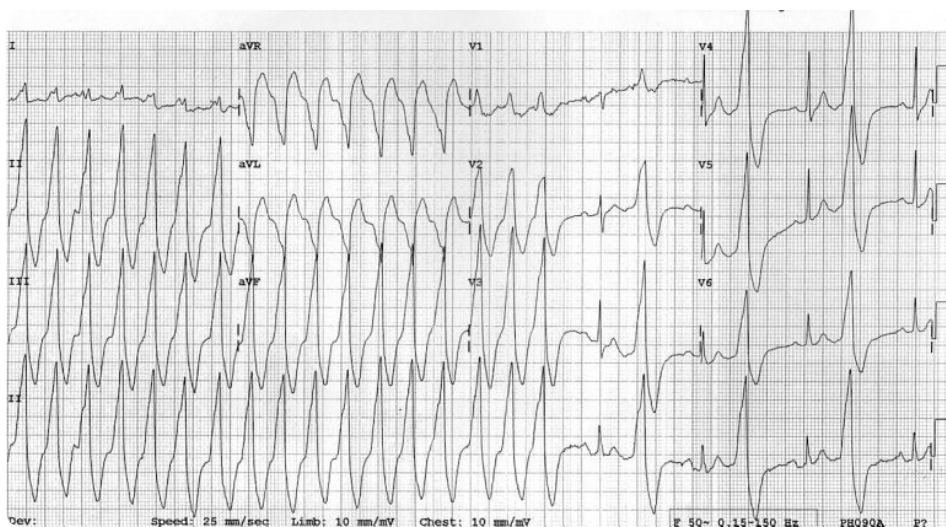
- **Ventricular Tachycardia**



- Supraventricular and a ventricular impulse coincide to produce a **hybrid complex**
- Indicates there are **2 foci** of pacemaker cells firing simultaneously



- The return of **atrial control** over ventricular contraction, following a period of atrioventricular dissociation



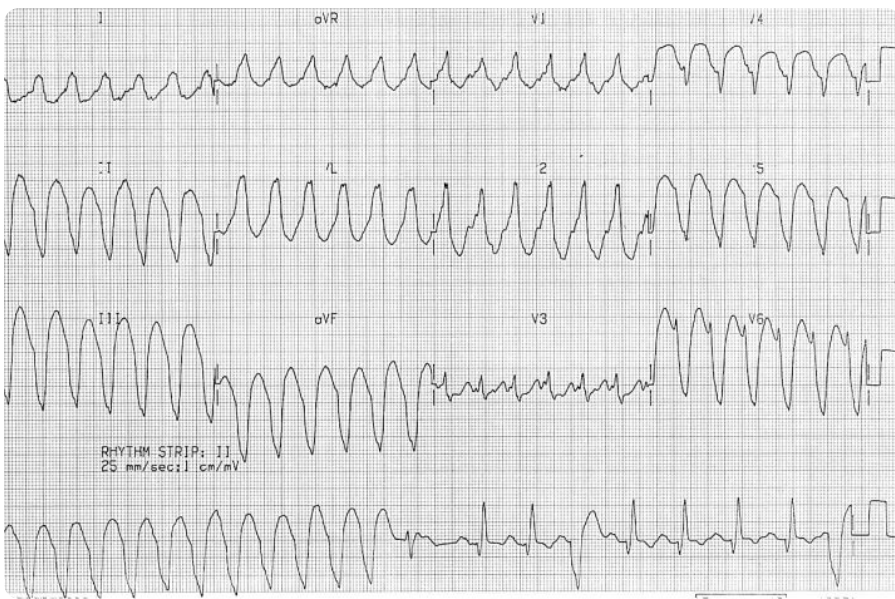
• **WIDE QRS COMPLEX**

• **FUSION BEATS:**

- occurs when a sinus & ventricular beat coincide & produce a hybrid complex

• **CAPTURE BEATS:**

- occurs when SAN transiently captures the ventricle, in the midst of AV dissociation to produce **Normal DURATION QRS Complex**.



• WIDE QRS COMPLEX

• FUSION BEATS

• CAPTURE BEATS

↓
VENTRICULAR
TACHYCARDIA

NOTE: AXIS OVER HERE
NORTH WEST
as aVR is positive

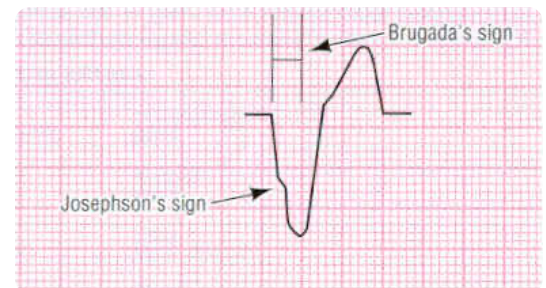
JOSEPHSON'S SIGN & BRUGADA'S SIGN

JOSEPHSON'S SIGN :

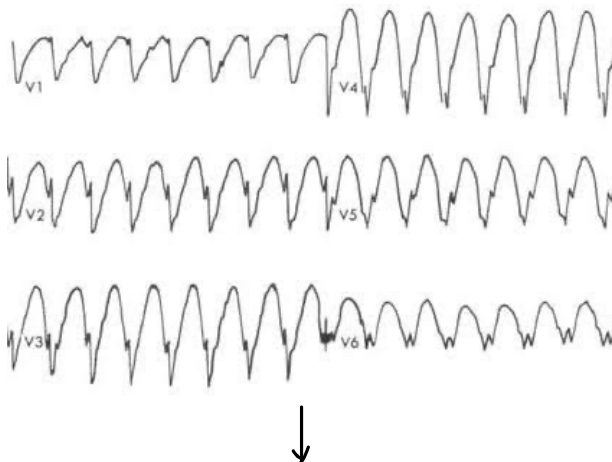
- Notching near the nadir of S-wave

BRUGADA'S SIGN :

- The distance from the onset of QRS Complex to nadir of S wave
- It's **>100ms**

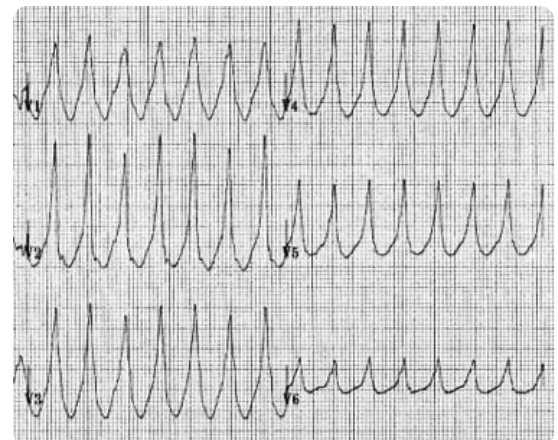


NEGATIVE & POSITIVE CONCORDANCE



↓
NEGATIVE CONCORDANCE

V₁-V₆ contain negative waves



↓
POSITIVE CONCORDANCE

V₁-V₆ contain positive waves

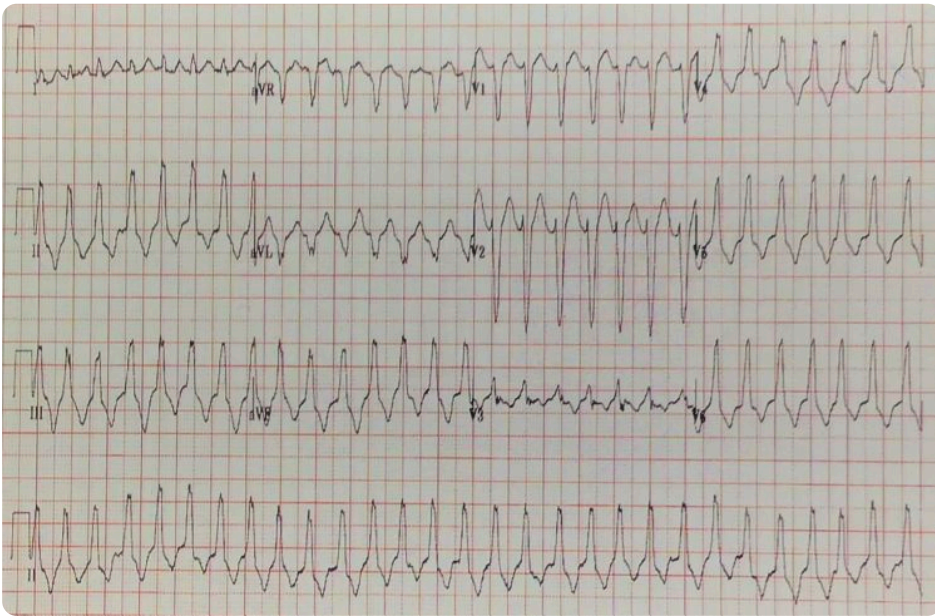
- In VT either positive or negative concordance present.

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KEY POINTS OF VT

1. WIDE QRS TACHYCARDIA ($>160\text{ms}$)
2. POSITIVE/NEGATIVE CONCORDANCE
3. AV DISSOCIATION
4. CAPTURE BEATS
5. BRUGADA'S SIGN
6. JOSEPHSON'S SIGN
7. MOSTLY NORTH-WEST AXIS : I, II, III is inverted
(Extreme axis deviation) aVR is upright
8. Absence of typical RBBB or LBBB morphology

Rate $>100\text{beats/min}$

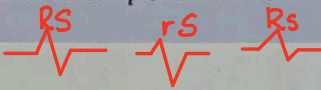


- WIDE QRS COMPLEX
 - REGULAR RR INTERVAL
 - NO CONCORDANCE
 - AV DISSOCIATION
- ↓
- VENTRICULAR TACHYCARDIA

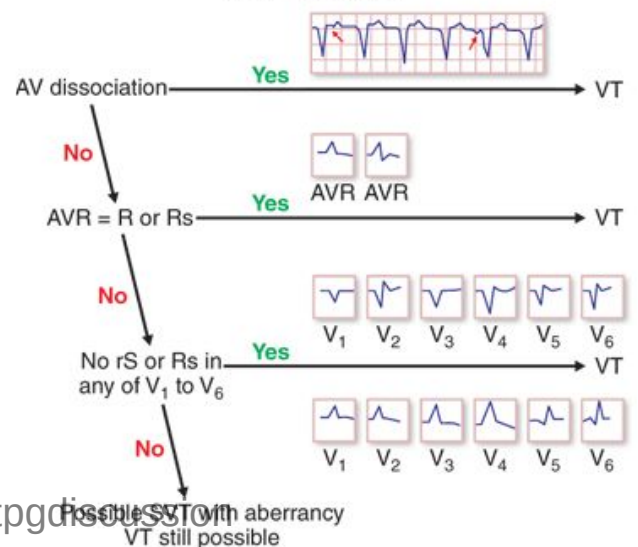
BRUGADA CRITERIA

TO DETERMINE whether it's SVT with aberrancy or VT.

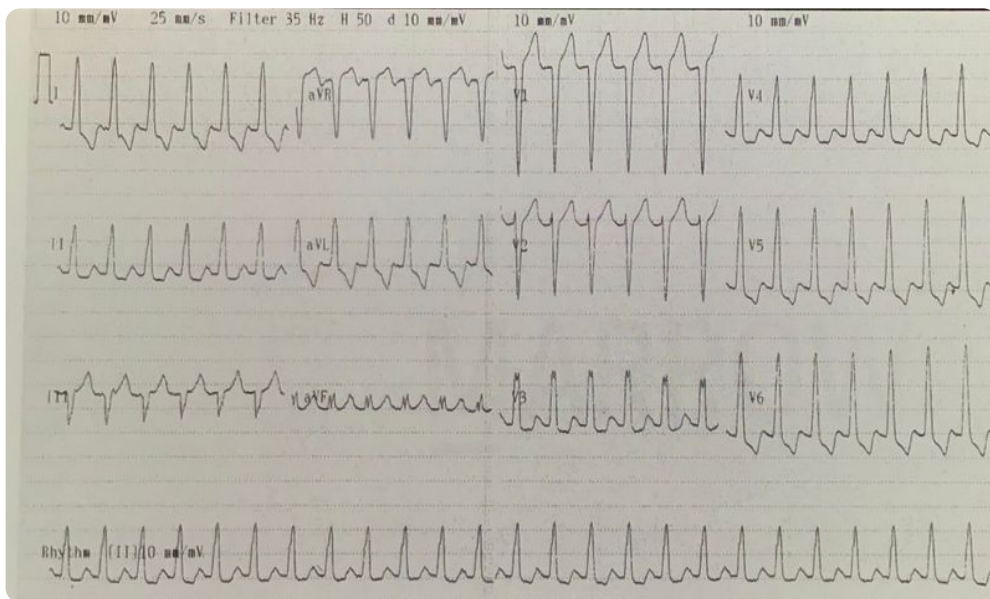
	YES
Absence of RS complex in $V_1 - V_6$	VT
RS complex duration $>100\text{ms}$	VT
AV dissociation	VT
Morphology criteria	VT



VT versus Supraventricular Tachycardia with Aberrancy



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Rate >200 beats/min

- NARROW QRS COMPLEX
(as VT is >160ms)
- LBBB Morphology
- No P waves

↓
AVNRT with LBBB Morphology

MANAGEMENT OF VT

- Generally Patients are Hemodynamically unstable

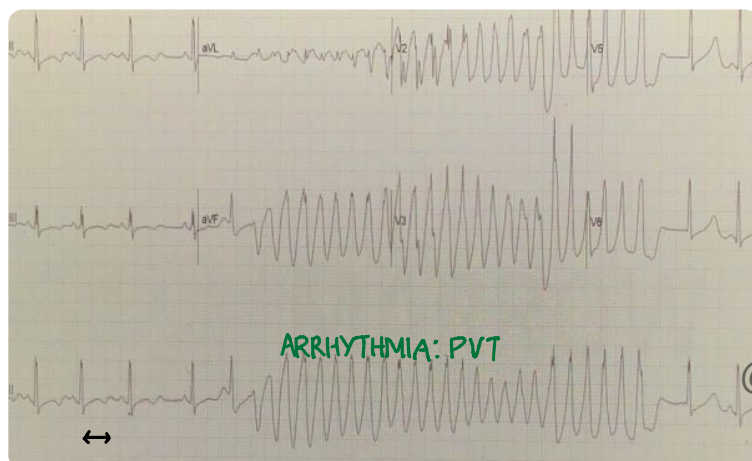
↓
TOC: ∴ Biphasic DC Cardioversion (100-360J) (MC: 200J)

- if patient is HEMODYNAMICALLY STABLE

- 1. **PROCAINAMIDE** in monomorphic VT & good LV function
- 2. **AMIODARONE** in LV Dysfunction
- 3. **LIGNOCAINE** in MI

VENTRICULAR TACHYCARDIA - POLYMORPHIC

- When different QRS Complexes of Different heights : Polymorphic
- Typically associated with **TORSADE DE POINTES**
- Polymorphic VT + PROLONGED QT INTERVAL = **TORSADE DE POINTES**



- Prolongation of QT interval

↓
Trigger an arrhythmia
(Polymorphic VT)

↓
This is called **TORSADE DE POINTES**

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CAUSES OF QT PROLONGATION

- CONGENITAL LONG QT SYN.

a) Long QT Syndrome Type 3:
due to Mutation in SCN5A gene

↓
in Brugada syn.

c) Jerwell Lange Nielson Syndrome
is associated with sensory
neuro hearing loss with QT
prolongation.

d) Romanowd Syndrome

- ACQUIRED QT PROLONGATION

a) Most important cause : Drugs

: CLASS IA AAD
CLASS II AAD
ANTIBIOTICS : MACROLIDES, CLINDAMYCIN
ANTI(FUNGAL) : KETOCONAZOLE
ANTIVIRAL : AMANTIDINE
ANTI PSYCHOTICS: HALOPERIDOL, TCS
ANTI HISTAMINE: TERFENADINE, ASTEMIZOLE,
FEXOFENADINE

b) CARDIAC CONDITIONS

: MI

c) ENDOCRINE DISORDER

: HYPO THYROIDISM
HYPER PARATHYROIDISM

d) INTRACRANIAL DISORDER

: CNS BLEED/INFARCTION

e) ELECTROLYTE ABNORMALITY

: HYPO Ca^{2+}
HYPO K^+
HYPO Mg^{2+}

f) HYPOTHERMIA

NOTE:

HYPERCALCEMIA } SHORTEN
DIGOXIN } QT
HYPER THERMIA } INTERVAL

TREATMENT OF POLYMORPHIC VT

- Mostly patients are HEMODYNAMICALLY UNSTABLE
TOC : DEFIBRILLATION

✓ if patient is hemodynamically STABLE & ACQUIRED QTSYNDROME

i/v Mg^{2+} → TEMPORARY PACING
Then

- As it can re occur : IMPLANTABLE CARIOVERTOR DEFRILLATOR

✓ if patient is hemodynamically STABLE & CONGENITAL QT SYNDROME

DOC : BETA BLOCKERS

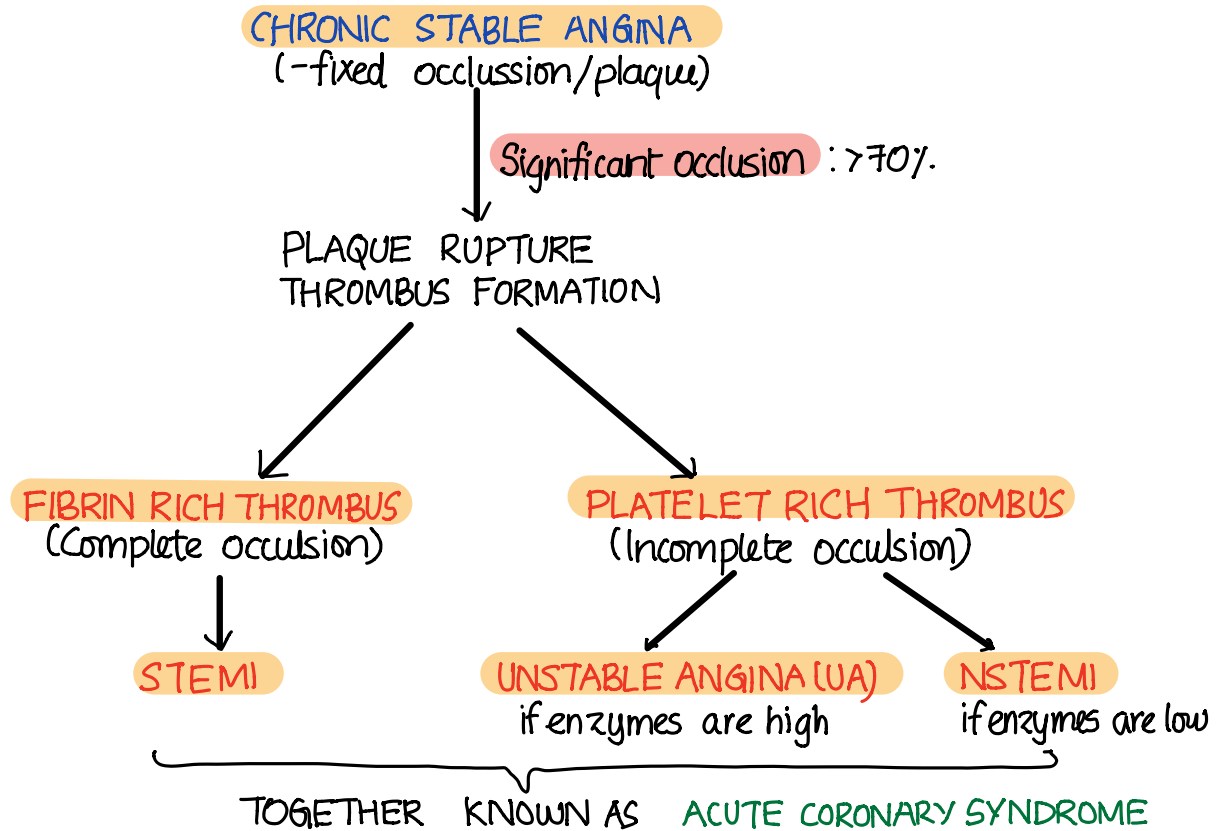
TABLE 277-3 CAUSES OF QT PROLONGATION AND TORSADE DE POINTES VENTRICULAR TACHYCARDIA

1. Congenital long QT syndromes (see text for details)	Antihistamines (histamine 1-receptor antagonists)
Long QT syndrome type 1: Reduced repolarizing current I_{Kr} due to mutation in <i>KCNQ1</i> gene	Terfenadine, astemizole, diphenhydramine, hydroxyzine
Long QT syndrome type 2: Reduced repolarizing current I_{Kr} due to mutation in <i>KCNH2</i> gene	Cholinergic antagonists: Cisapride, organophosphates
Long QT syndrome type 3: Delayed inactivation of the I_{Na} due to mutations in <i>SCN5A</i> gene	Citrate (massive blood transfusions)
Others: Several other types of long QT syndromes have been described; long QT types 1, 2, and 3 account for 80–90% of cases	Cocaine
2. Acquired prolongation of QT interval	Methodone
Electrolyte abnormalities	Fluoxetine (in conjunction with other drugs that prolong QT)
Hypokalemia	Cardiac conditions
Hypomagnesemia	Myocardial ischemia and infarction
Hypocalcemia	Myocarditis
Drugs	Marked bradycardia
Antiarrhythmic drugs	Stress cardiomyopathy
Class IA: Quinidine, disopyramide, procainamide	Endocrine disorders
Class III: Sotalol, amiodarone (QT prolongation common but torsade ventricular tachycardia is rare), ibutilide, dofetilide, almokalant	Hypothyroidism
Antibiotics	Hyperparathyroidism
Macrolides: Erythromycin, clarithromycin, azithromycin	Hyperaldosteronism
Fluoroquinolones: Levofloxacin, moxifloxacin, gatifloxacin	Intracranial disorders
Trimethoprim-sulfamethoxazole	Subarachnoid hemorrhage
Clindamycin	Thalamic hematoma
Pentamidine	Cerebrovascular accident
Chloroquine	Encephalitis
Antifungals: Ketoconazole, itraconazole	Head injury
Antivirals: Amantadine	Nutritional disorders
Antipsychotics	Anorexia nervosa
Haloperidol, phenothiazines, thioridazine, trifluoperazine, sertindole, zimelidine, ziprasidone	Starvation
Tricyclic and tetracyclic antidepressants	Liquid protein diets
	Gastroplasty and ileocejeunal bypass
	Celiac disease



ACUTE CORONARY SYNDROME

PART - I

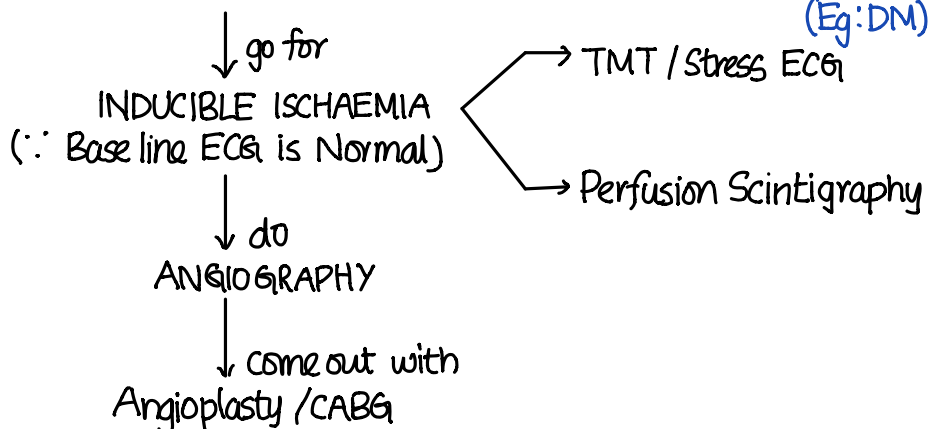


2/3 CRITERIA FOR ACS : 2 out of 3 Criteria are mandatory to diagnose ACS

- ① SYMPTOMS ② ECG ③ ENZYME

AIMS

1. TO PICK UP PATIENT WITH CHRONIC STABLE ANGINA OR HIGH RISK PATIENTS (Eg: DM)



2. TO PICK PATIENT WITH ACUTE ACS
- CLASSIFY @neetpgdiscussion
- STEMI
 - NSTEMI
 - UA

RISK FACTORS

- Family history of MI
- Chronic Stable Angina : Angina on exertion (or) Dyspnea on Exertion
- **CIGARETTE SMOKING**: MOST CRUCIAL YET PREVENTABLE RISK FACTOR
 - ↓ do a Angiogram
 - * Smoking 5-6 packet/day for 20 years (or) 2-3 packet/day for 10-12 years
 - * Also Diffuse Vascular Disease present. As it effect
 - ILLIAC VESSAL
 - LOWER LIMB VESSAL
 - RENAL ARTERY
 - CORONARY ARTERY
 - Caliber of vessal is lost
 - ∴ **DISTAL TAPPERING** of BV (You can't stent the vessal)
- **HYPERTENSION** (BP $\geq 140/90$)
- HIGH LDL

(i) Highly Sensitive CRP : DFFINITE	(ii) Apo B100/HDL Ratio
MARKER OF ATHEROSCLEROSIS	
→ Lp(a) Level also	(iv) Total cholesterol/HDL Ratio
(iii) Fibrinogen level	(vi) LDL/HDL Ratio
(v) TG/HDL Ratio	

- LOW HDL (Most important : HDL-2)
- DIABETES MELITUS
 - * leads to **INSULIN RESISTENCE**
- IN WORLD MALES HAVING MORE RISK THAN FEMALE

→ Male > 45 yrs
Female > 55 yrs

IN INDIA

- Age of presentation : 45-50 YRS
- **FEMALE** > **MALE**
 - ↓ ACS present as ATYPICAL PRESENTATION
 - ↓ ACS present as CHEST PAIN
- OBESITY
 - But IN INDIA: Even Pt with NORMAL BMI : Risk of ACS
- MALNUTRITION, INFLAMMATION, ATHEROSCLEROSIS
- HOMO CYSTENEMIA
- METABOLIC SYNDROME

Waist Circumference : 120/88
TG : 150
BP : 130/85
FBP : 100
HDL : <50 / <40



✓ All these patient should go for
 → STRESS ECG
 → PERFUSION SCINTIGRAPHY

not for Resting ECG : It's normal in these patient
 (or) Echocardiogram : Structurally the heart is normal
 Sometimes Diastolic dysfunction in some patient

✓ 5-6 patients out of 10 ACS patient develop Heart failure because of INFARCTION
 This is called as ISCHAEMIC DILATED CARDIOMYOPATHY

INDICATIONS TO PUT PATIENTS ON STATINS

1. Clinical Atherosclerosis evidence
 2. DM (40-75 yrs age)
 3. LDL > 100 mg/dl
- } MODERATE TO HIGH DOSE STATIN
 40mg ATORVASTATIN (or)
 20mg ROSUVASTATIN

- AS STATIN : NO EFFECT ON HDL
 NO EFFECT ON TG
 ONLY EFFECT ON LDL

- MOST Patients have DYBETIC DYS LIPIDEMIA
 due to ↓ HDL
 ↑ TG

MOST ATHEROGENIC
 ↑ small dense/ oxidised LDL

} NO EFFECT BY STATINS
 ∴ SAROGILITAZAR

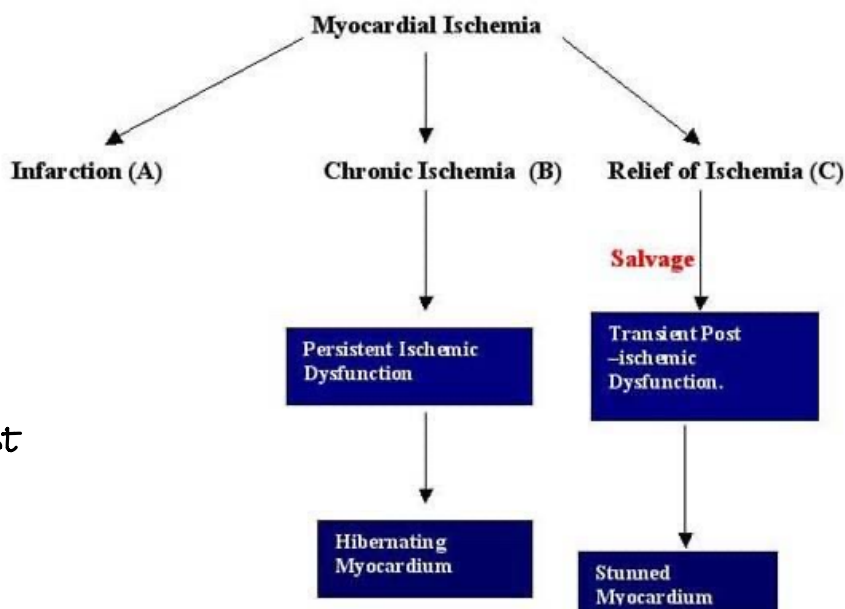
HIBERNATING MYOCARDIUM & STUNNED MYOCARDIUM

HIBERNATING MYOCARDIUM

- Areas of myocardium which are persistently under perfused but still viable after re perfusion
- Occurs in chronic ischaemia
- Reversible

STUNNED MYOCARDIUM

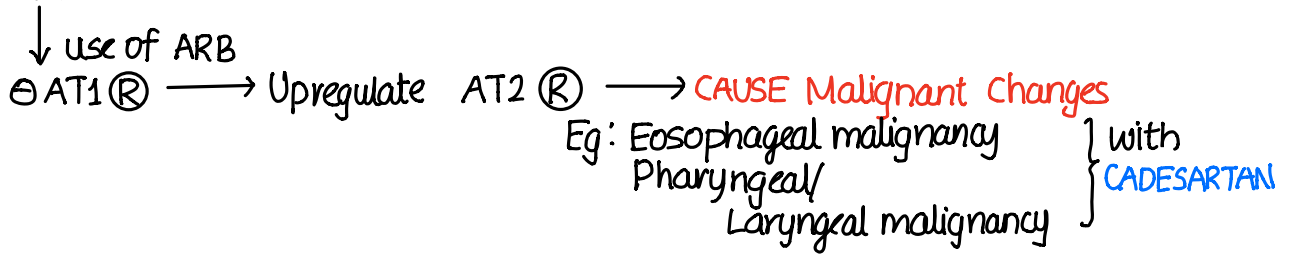
- Segmental dysfunction which persist for a variable period of time after reperfusion is called STUNNING
- Occurs in Acute ischaemia.
- Reversible



NOTE:

- **HOPE Trial** Demonstrates that **RAMIPRIL** reduces fatal & non fatal vascular events in high risk patients

- On Target study reveals that ARBs are not inferior to ACE $\ominus$



- CONTRAINDICATIONS FOR ACE $\ominus$

1. PREGNANCY
2. h/o HEREDITARY ANGIONEUROTIC EDEMA
3. b/i Renal Artery Stenosis

Note : CREATININE Level is not a C/I

- AFTER starting ACE $\ominus$ Monitor for Creatinine level

① if $>30\%$ increase in creatinine level after starting ACE $\ominus$ } STOP ACE $\ominus$

②. HYPER KALEMIA : if it's $>5.5-6$ within 1wk. : STOP ACE $\ominus$

DIETARY/LIFESTYLE RECOMMENDATION

- Saturated Fat $<7\%$
- Dietary Cholesterol of $<200\text{mg/day}$
- Soluble fibre $>10-25\text{g/day}$
- Plant sterols : 2g/day
- $>15\%$ protein
- Salt less than 6g/day
- No trans fatty acids

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Message @drnash1 for group discount upto 50% on any lecture app and individual lecture app.

ACUTE CORONARY SYNDROME

SPECTRUM OF PATIENTS

1. HEART FAILURE :
 - Systolic HF
(Most often by patient who are already developed MI)
 - Diastolic HF
2. CHRONIC STABLE ANGINA : Occurs during exertion <20 minute duration
3. Unstable Angina / NSTEMI : Occurs at Rest, duration >20 minutes
4. STEMI
5. PRINZMETAL'S ANGINA / VASOSPASTIC ANGINA : Occurs at rest <20 minutes duration

CHRONIC STABLE ANGINA

- Defined by
 - i) Precordial pain precipitated by stress or exertion
No pain at rest
May be tightness / burning / squeezing
Behind or to left of midsternum with radiation to any dermatome from C8 to T4
 - ii) Duration less than 20mnts ...
If more than 30mnts suspect ACS
 - iii) Excellent response to NTG
- ACUTE MR in chronic stable angina : **Diagnosis: Papillary Muscle dysfunction**

EXERCISE ECG / STRESS ECG / TREAD MILL TEST

- Recommended initial procedure
- It's according to **BRUCE PROTOCOL**
- **C/I** : * Rest Angina
* Symptomatic Aortic stenosis
* Pt who donot want to do the test : DON'T PUSH.
- Significant ST Down slopping : * 2mm in 6mnts before achieving the max HR are considered to be very significant
- * if we keep the cut-off as 1mm : 30% chance of false positivity

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MYOCARDIAL PERFUSION SCINTIGRAPHY

- It's also called called **Perfusion scan**

↓
USE : Thallium

- INDICATION:

- * Symptoms at Rest
- * Positive test in asymptomatic patient

- ADVANTAGE

- * Localize region of ischaemia
- * Differentiate Ischaemia & Infarction
- * Assess completeness of revascularisation

- ALTERNATIVE : **Exercise Radionuclide Angiography / MUGA Scan**

- * **Perfusion Scintigraphy/MRI** can distinguish stunned myocardium from Scarred Myocardium
- * **Electron beam CT (EBCT) TO QUANTIFY CARDIAC CALCIFICATION**
- * **MRI** : Test of Choice to IDENTIFY MYOCARDIAL FIBROSIS
- * **AGATSTON SCORE** : IS BASED ON EXTENT ON CORONARY ARTERY CALCIFICATION
- * **70% OR MORE** narrowing associated with ischaemia
50% or More is significant
- * **INTRAVASCULAR ULTRASOUND** : Test of Choice for Ostial left main lesion & coronary dissection

MANAGEMENT

- S/L NTG : Action within 2-5 mins.
0.3-0.6 mg
3 tablets within 20 min

↓ Not Responding

UA/NSTEMI/STEMI

- **Nitrate Tolerance** is the main limitation of NTG

→ To prevent : keep Nitrate free interval 8-12 hrs period daily

- **Beta Blockers** : Antianginal drug with mortality benefit

- **Ranolazine** : PROLONG EXERCISE DURATION & TIME TO ANGINA

→ S/E : **QT Prolongation**

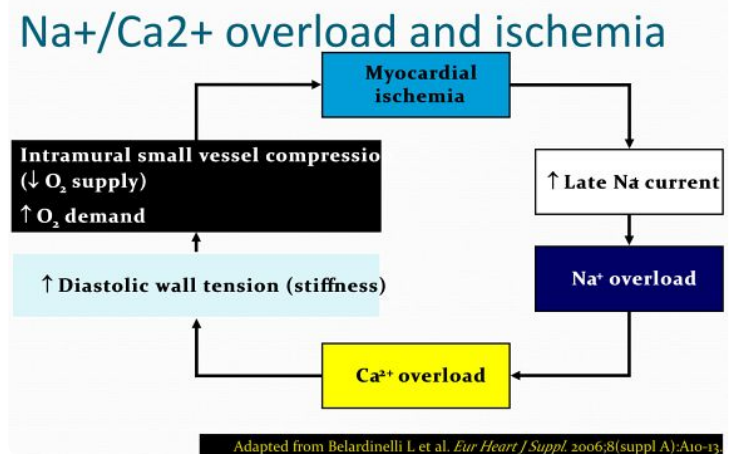
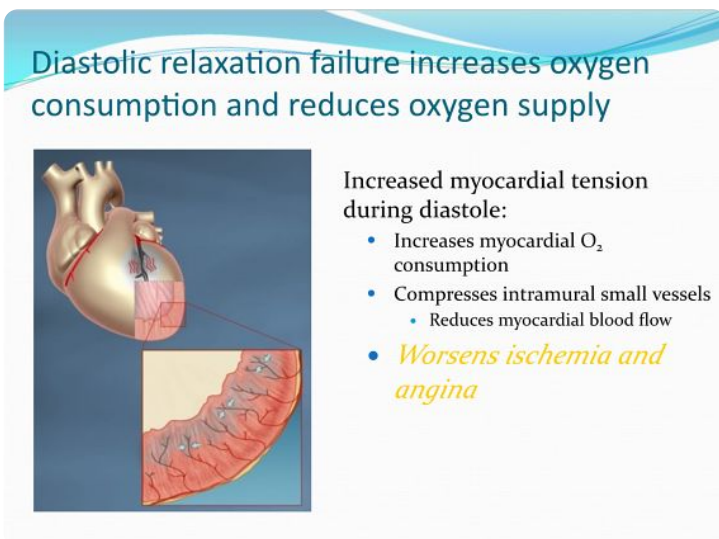
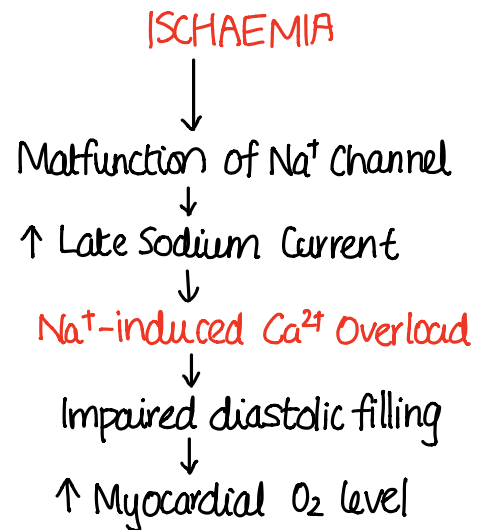
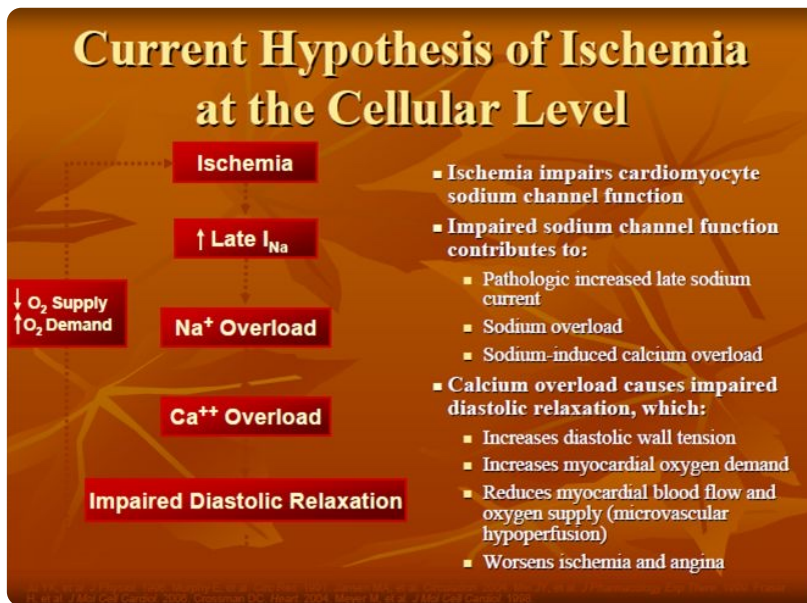
Oral ISDN : Take 30 min
Oral ISMN : Take 45 min

- **IVABRADINE** : Blocks Funny current $\rightarrow$ $\downarrow$ H.R

- CCBs are third line drugs

DRUGS IN ANGINA

- * First Line : NTG
- * Second Line : Beta blockers
Ranolazine
Ivabradine
Nicorandil
- * Third line : CCBs
Trimetazidine



Reduce E.D.V	$\Rightarrow$	$\downarrow$ O ₂ Consumption	: Nitrate
$\downarrow$ H.R	$\Rightarrow$	$\downarrow$ O ₂ Consumption	: β ⊖, CCBs
pFOX ⊖	$\Rightarrow$	$\downarrow$ O ₂ Consumption	: Ranolazine
Rhokinase ⊖	$\Rightarrow$	$\downarrow$ O ₂ Consumption	: Fasudil
SAN ⊖	$\Rightarrow$	$\downarrow$ O ₂ Consumption	: Ivabradine

TRIMETAZIDINE / RANOLAZINE

- These are p-FOX $\ominus$

S/E: QT Prolongation

$\ominus$ β oxidation of FFA ($\uparrow$ ATP utilisation)

Myocyte utilise the Glucose ($\downarrow$ ATP utilisation)

$\downarrow$ O₂ Requirement

- At the time of ischaemia use FA oxidation

IVABRADINE

- M.O.A: I_f current $\ominus$
(funny current)

$\ominus$ SA Node

$\downarrow$ H.R

NICORANDIL

- M.O.A: ACTIVATION OF ATP SENSITIVE K⁺ CHANNELS

Dilatation of Resistance Vessels

FASUDIL

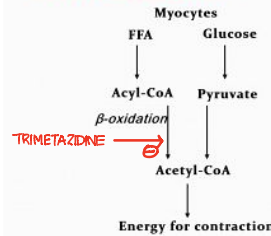
M.O.A: Rho Kinase Inhibitor

Summary

Medication Class	Impact on HR	Impact on BP	Physiologic Mechanism
Beta Blockers	$\downarrow$	$\downarrow$	Decrease pump function
Calc Channel Blockers	$\downarrow$	$\downarrow$	Decrease Pump function + Vaso-dilatation
Nitrates	$\uparrow$	$\downarrow$	Vaso-dilatation <i>Reflux tachy cardia</i>
Ranolazine	$\bigcirc$	$\bigcirc$	Reduced Cardiac Stiffness

Metabolic modulation (pFOX):

Trimetazidine



pFOX = partial fatty acid oxidation
FFA = free fatty acid

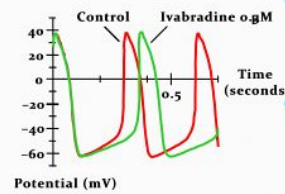
- O₂ requirement of glucose pathway is lower than FFA pathway
- During ischemia, oxidized FFA levels rise, blunting the glucose pathway

MacInnes A et al. *Circ Res* 2003;93:e26-32.

Lopaschuk GD et al. *Circ Res* 2003;93:e33-7.

Stanley WC. *J Cardiovasc Pharmacol Ther* 2004;9(suppl 1):S31-45

Sinus node inhibition: Ivabradine

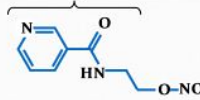


- I_f current is an inward Na⁺/K⁺ current that activates pacemaker cells of the SA node
- Ivabradine
 - Selectively blocks I_f in a current-dependent fashion
 - Reduces slope of depolarization, slowing HR

Preconditioning: Nicorandil

Activation of ATP-sensitive channels

- Ischemic preconditioning
- Dilation of coronary resistance arterioles

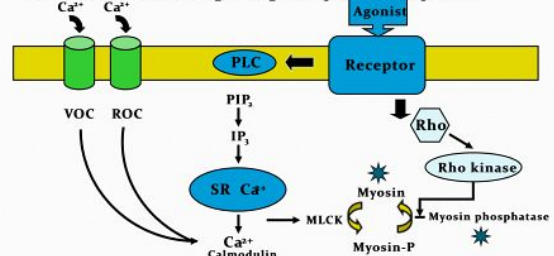


Nitrate-associated effects

- Vasodilation of coronary epicardial arteries

Rho kinase inhibition: Fasudil

- Rho kinase triggers vasoconstriction through accumulation of phosphorylated myosin



@neetpgdiscussion

- Q: Patient with Precordial chest pain during rest without precipitant rapidly relieved by Nitrates, Transient ST Elevation on ECG. Normal Angiography.
Nitrate/CCB useful but not preferred
- * Chronic pattern of recurrent episodes of chest pain
 - * Episodes midnight to early morning
 - * Episode last for 5 to 15 minutes
 - * Physical Examination: No finding characteristic to VA. However during episode Tachycardia, HTⁿ, Diaphoresis & a gallop rhythm present

Diagnosis : PRINZ METAL'S ANGINA / VASOSPASTIC ANGINA .
(due to transient vasospastic episodes)

- Seen in smokers
- Good Prognosis

ACUTE CORONARY SYNDROME

NSTEMI

- DEFINITION:

Rest angina > 20 minutes
New ONSET SEVERE ANGINA
CRESCENDO PATTERN

if this patient have
* ST Depression
* Enzymes +

NSTEMI

If this patient have
* ST depression
* Enzymes normal

UNSTABLE ANGINA

- According 2017 classification both together called as NSTEMI-ACS

- TIMI RISK SCORE : Determine mortality risk in NSTEMI

MANAGEMENT

1. S/L NTG : 0.3-0.6 mg x 3 times within 20 minutes

↓ NOT RESPONDED

Give MORPHINE

2. ASPIRIN : - Essential drug in ACS, STEMI & NSTEMI
- For 1^o PCI or URGENT setting

Loading dose : 325 mg/day (non-enteric coated)

- HARRISON : Hospital dose : 80-325 mg/day

- Higher doses ↑ bleeding risk without additional benefit

- Every cases of CAD without c/I should be prescribed ASPIRIN

- Maintenance Dose : 75 mg

3. IN EVERY CASE OF NSTEMI-ACS : Should combine
CLOPIDOGREL X ASPIRIN

Continued for 12 months
Aspirin maintenance dose <100mg

Continue ASPIRIN for Life
CLOPIDOGREL for 12 months

Aspirin x ADP Receptor Antagonist

1. CLOPIDOGREL : Loading dose : 300mg
Maintenance : 75 mg
For 1yr (12 month)

Drawback :

- Show CYP2C19 Polymorphism
↳ Activator of Clopidogrel

2. PRASUGREL : - 10 times more potent
- Rapid onset of action
- Better absorption
- But still CYP2C19 activation

3. TICAGRELOR
• LD: 180mg
• MD: 90mg BD
• for 12 months
- No activation by CYP2C19
- Reversible inhibitor
- Short $t_{1/2}$ + ORAL
- S/E : Mild Dyspnoea

4. STATINS : - In First 24hrs
• Risk for UA in the next 4 month reduces by 25%
Atorvastatin : 40-80mg

5. GP_{IIb-IIIa} ANTAGONIST :

ABCIXIMAB : PLASMA $t_{1/2}$ Short ; Platelet bound $t_{1/2}$: Long
SAFE IN RENAL FAILURE

EPTIFIBATIDE : LONGEST $T_{1/2}$
TIROFIBAN

ROLE : ↓ PERIPROCEDURAL MI (MI developed during PCI)

6. HEPARIN : - 33% Reduction in death in 1st week
- Major Reduction in non-fatal MI
- NO SIGNIFICANT INCREASE IN BLEEDING
- NO DECREASE IN RECURRENT ANGINA OR
REVASCULARISATION PROCEDURE

DRUGS : UNFRACTIONATED HEPARIN :

LMWH

FONDAPARINUX

DIRECT THROMBIN INHIBITORS : Eg: DABIGATRAN

@heetpdiscussion

ENOXAPARIN & FONDAPARINUX is preferred

ENOXAPARIN (LMWH) : Advantages over Unfrac. Heparin

↓
for 7-8 days

1. No inhibition of platelet function
2. No Thrombocytopenia
3. NO LIVER TOXICITY
4. Patient variability less

✓ CLASS I INDICATION: ASPIRIN
CLOPIDOGREL
UFH / ENOXAPARIN

- ✓ For initial conservative strategy
 - to prevent Periprocedural MI
 - Indication if pt had Recurrent Angina
 - ABCIXIMAB should not be used

} EPTIFIBATIDE
TIROFIBAN } GP IIb-IIIa[⊖]

KILLIP CLASSIFICATION

Killip Subgroup	Clinical characteristics	Hospital mortality
I	No congestion signs	<6%
II	S3, basal rales	<17%
III	Acute pulmonary edema	38%
IV	Cardiogenic shock	81%
Forrester subgroup	Hemodynamic characteristics	Hospital mortality
I	PCP <18, IC >2.2	3%
II	PCP >18, IC >2.2	9%
III	PCP <18, IC <2.2	23%
IV	PCP >18, IC <2.2	51%

PCP- pulmonary capillary pressure; CI- cardiac index.

• Predicting mortality by accessing LV function

• CLASS I : NO SIGN OF LV FAILURE

• CLASS II : S3, BASAL RALES

• CLASS III : PUL. EDEMA

• CLASS IV : CARDIOGENIC SHOCK

• IN LV Failure take to IABP (Intra aortic Balloon Pump)
↓ followed by Angiogram

• JVP Elevation
Clear Lungs
Hypotension } CASE OF RV FAILURE
↓ t/t
Pump with fluids

FORRESTER & DIAMOND CLASSIFICATION

* It's modified Killip classification

Simplified Forrester & Diamond hemodynamic classification in STEMI		
Class 1	No hypotension	No pulmonary congestion
Class 2	No hypotension	Pulmonary congestion
Class 3*	Hypotension	No pulmonary congestion
Class 4 *	Hypotension	Pulmonary congestion

* Include RV and Bi-Ventricular MI. ** Cardiogenic shock . www.drsvenkatesan.co.in

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Q Triple Vessel disease

Severe LV Systolic Dysfunction

2 vessel disease with proximal LAD occlusion

Left main disease

Diagnosis : CABG

ACUTE MI

- Evidence of Myocardial necrosis in the settings of Ischaemia

↓ i.e

Rise of cardiac biomarkers (Mostly Troponin)
followed by any of the following

1. New ST/T Changes or New LBBB
2. Symptoms of ischaemia
3. Pathological Q waves on ECG
4. Evidence of thrombus by angiogram
5. Impaired perfusion on scintigraphy or new Regional Wall Motion anomaly on Echo

- BIOMARKERS

• FIRST MARKER TO RISE : MYOGLOBIN

• LAST MARKER TO RISE : LDH

• AST : Liver/Skeletal muscle/ Coronary specific
ALT : Liver specific



∴ ↑ AST & ⊕ ALT → Non hepatic causes : • Rhabdomyolysis
• MI

Liver : ALT > AST Except : * Alcohol Hepatitis (∴ def Pyridox.phos)
* Cirrhosis

• CPK : - seen at : 2-4hrs
Peak : 24hrs
Normalise : 48-72 hrs
- Advantage : * Almost first to rise

* High sensitivity

* In the setting of Reinfarction

• TROP-T : - MOST SPECIFIC

- Begins : 4-6 hrs

- Peak : 48 hrs

- Normalise : 7-10 days (if Trop-I : 14 days)

TROP-I : USED IN RENAL FAILURE



- CLASSIFICATION OF MI

- CLASS I : Spontaneous MI (Routine MI)
- CLASS II : MI due to Non CAD Causes due to ischaemic imbalance
- CLASS III : MI causing death blood samples unavailable
- CLASS 4a : MI Relating PCI
- CLASS 4b : MI Relating to Stent thrombosis
- CLASS 5 : MI Relating to CABG

- MANAGEMENT

- i) NTG (3 tab) NOT RESPON. → MORPHINE
- ii) Aspirin 325mg
Ticagrelor 180mg / Clopidogrel 300mg
- iii) Atorvastatin 40-80 mg
- iv) Anticoagulant (LMWH / FONDAPARINUX)
- v) Planning for procedure GPIIb/IIIa ⊖

C/I of NITRATES

- * SEVERE HYPOTENSION
- * WITH SILDENEFIL

2 MORE DRUGS TO START IN FIRST 24 HRS

- vi) BETA BLOCKERS - C/I : AV BLOCK
LV FAILURE
BRADYCARDIA (<60)

vii) ACE INHIBITORS

SALVAGING OF MYOCARDIUM : THROMBOLYSIS v/s PRIMARY PCI

• GOLDEN HOUR:

The benefit of intravenous thrombolysis is extremely time sensitive and when treatment is established in the first 2hr after symptom onset, the so called **GOLDEN HOUR**, survival dramatically decreased.

i.e **THROMBOLYSIS** have survival benefit if initiated within 2 hrs.

- | | | |
|------------------------|---|----------------------------|
| if it's within 30 mnts | → | AS GOOD AS PCI |
| if it's within 1 hr | → | 90% as good as PCI |
| if it's within 2 hr | → | Definite survival benefit. |
| Can be done up to | → | 12 hrs |
| >12hr | → | No benefits |

- PCI is superior to Thrombolysis
- In PCI

DOOR TO BALLOON TIME : <90 minutes

if you are transferring : <120 minutes

- Usually Thrombolysis soon followed by PCI
- It's associated with severe bleeding

FACILITATED PCI



Notespaedia.com
More than just notes...

• PHARMACOLOGICAL APPROACH :

Do Thrombolysis at first possible point



And do PCI Later (3-24 hrs Late) with full dose of
HEPARIN / GP IIb/IIIa

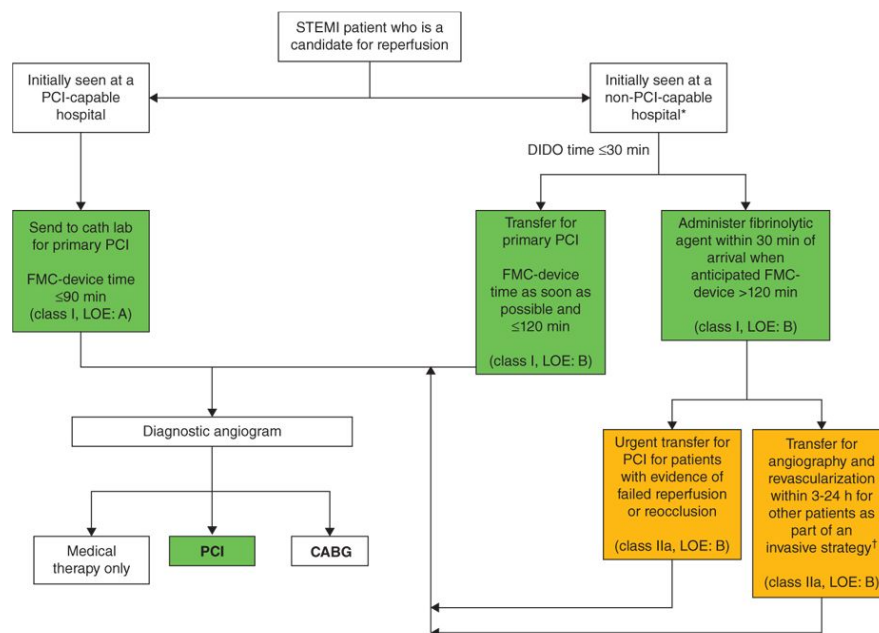
FACILITATED PCI

1. PCI Capable Hospital
2. Decision for PCI taken + Pharmacological
3. PCI Done within 120 mnts
4. Antiplatelet ±
5. Prothrombotic effects
6. Fearing Bleeding

PHARMACOLOGICAL PCI

1. Admitted to Non PCI Hospital
2. PCI is back up after Lysis
3. PCI done after 3 hrs
4. Adequate antiplatelet.
5. Prevent occlusion
6. No risk of bleeding

• **RESCUE PCI** : Done in failed Thrombolysis . Not preferred .



Source: Habib Samady, William F. Fearon, Alan C. Yeung, Spencer B. King III: Interventional Cardiology, 2nd Edition Copyright © McGraw-Hill Education. All rights reserved.

THROMBOLYSIS

- **DOC : TENECTEPLASE** : Advantages →
 1. FIBRIN SPECIFICITY Very high
 2. FIBRIN AFFINITY High
 3. NO ALLERGIC REACTION
 4. SINGLE BOLUS ADMINISTRATION (0.5 mg/kg)
 5. Patency after 1 hr > 75%
 6. RISK OF BLEEDING LESS

→ ↑ Risk of ICH

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More than just notes...

- STREPTOKINASE : - 1.5 MU over 30-60min
 - RETEPLASE : - 10U over 2min
- ↓ ½ hour gap
- 10U over 2min

- ALTEPLASE

- C/I FOR THROMBOLYSIS

ABSOLUTE C/I

1. ACTIVE INTERNAL BLEEDING
2. INTRACRANIAL NEOPLASM
3. HEMMORRHAGIC CVA at any time
4. CVA/TIA within one year
5. Suspected Aortic dissection

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ECG CHANGES IN ACUTE CORONARY SYNDROME

SURFACES & BORDERS OF HEART

SURFACES

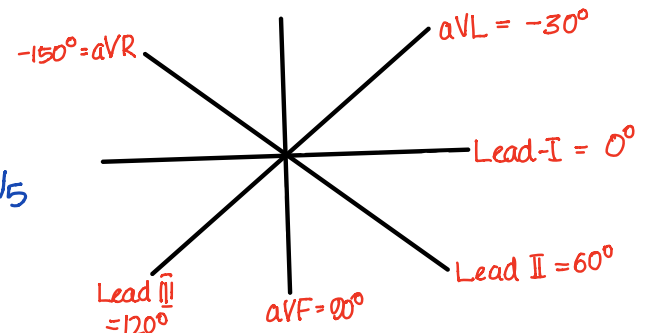
1. Anterior / Sternocostal : RV
2. Posterior : LA
3. Inferior / Diaphragmatic : RV+LV

BORDERS

1. Right border : RA
2. Left border : LV+LA
3. Superior border : LA+RA+ Aorta + Pulmonary trunk
4. Inferior border : RV+LV

LEADS

1. INFERIOR WALL : II, III, aVF
2. ANTERIOR WALL : V₂, V₃, V₄
3. SEPTUM : V₁, V₂
4. APEX : V₄ (Predominantly), V₅
5. RV FREE WALL : V_{4r}, V_{5r}, V_{6r}
6. POSTERIOR WALL : V₇, V₈, V₉
7. LATERAL LEADS : V₅, V₆
8. HIGHLATERAL LEADS : I, aVL



CIRCULATION

- RCA - in 45-50% : MC INVOLVED VESSEL
- LCA - in 36-40%
- LCX - in 10-15%

Right Coronary artery

- Start in the Right AV Sulcus

↓
Supplies SA Node

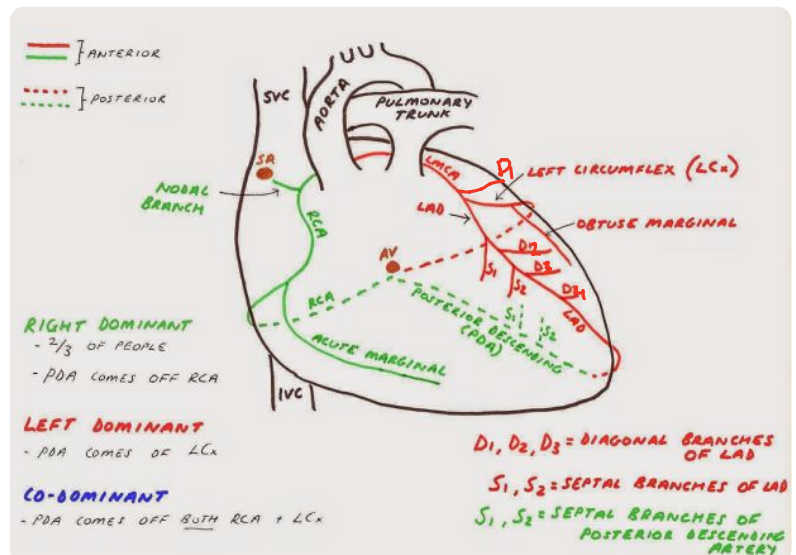
- a) ACUTE MARGINAL ARTERY
Supplies the RV Free wall

- b) Goes posteriorly & supply

Posterior Wall + Branch to AV Node

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- c) Posterior descending Artery : SUPPLY INFERIOR WALL OF HEART



✓ RV FREE WALL IS SUPPLIED ONLY BY : RCA

✓ POSTERIOR WALL } RCA/LCX or RCA & LCX
INFERIOR WALL } IN 85% RCA
15% LCX
RCA or LCX

✓ If the posterior descending artery is originating from — RCA : Right Dominant (in 85%)
LCX : Left Dominant (in 15%)
↓
Supply inferior wall

✓ If the patient have one proximal wall occlusion } RVMI + IWMI + PWMI (Post. Wall MI)

If the level of occlusion after marginal artery } IWMI + PWMI

If the occlusion is at PDA - Only IWMI (Inf. wall MI)

✓ COMPLICATION: a) Heart block : as it supply SA & AV Nodes
b) Atrial fibrillation
c) Pappillary muscle rupture —————> Acute MR
d) RVMI

NOTE:

Vagal afferent fibers can produce BRADYCARDIA by Reflux stimulation

So in an Inferior wall MI, Bradycardia may be due to

(i) Vessel occlusion

(ii) Reflux stimulation of vagal afferent

But in Anterior wall MI, Bradycardia denotes

EXTENT OF ISCHAEMIA

LEFT CIRCUMFLEX ARTERY

- Originate from Left Main Coronary artery (LMCA)

- Supplies

(i) Lateral wall of heart through OBTUSE MARGINAL ARTERY

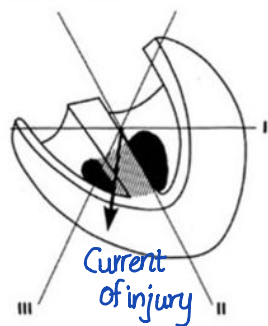
(ii) Posterior wall of heart

(iii) 10-15% cases Inferior wall of heart

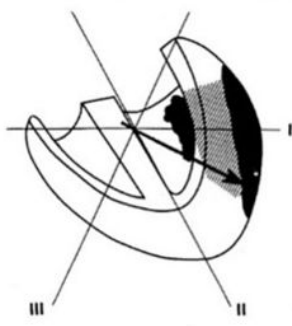
- NOTE: NO RVMI Here.



RIGHT CORONARY ARTERY MI



CIRCUMFLEX CORONARY ARTERY MI



NOTE:

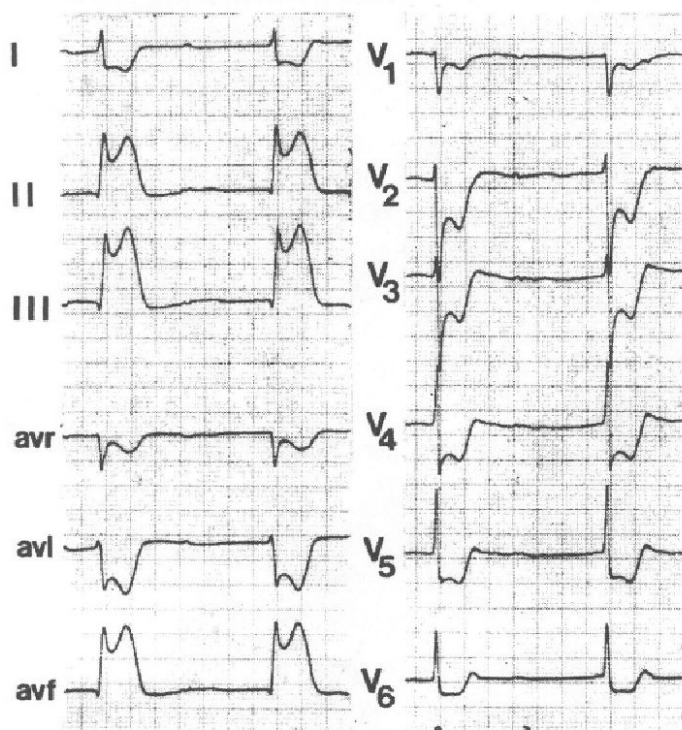
RCA Occlusion

ST Elevation in
Lead $\text{II} > \text{III}$

LCX Occlusion

ST Elevation in
Lead $\text{II} > \text{III}$

RIGHT CORONARY ARTERY



- LOOK FOR RECIPROCAL CHANGE
Here every lead got reciprocal change
i.e. it's ST ELEVATION

- ST Elevation in Lead II , III , aVF

Inferior Wall MI

RCA (in 85%)

Here ST Elevation is more in Lead $\text{III} > \text{II}$
∴ it's due to RCA

LCX (15%)

- As it's by RCA, check for

a) RVMi

On a 12 Lead ECG look for $V_1 - V_2$ In RVMi V_1 will show DISORDANCE with V_2
∴ Here RVMi also

b) PWMi

On a 12 Lead ECG look for V_2 In PWMi V_2 show ST Depression &
Reciprocal changes of ST Elevation
∴ Here PWMi

- Now find the Rate: Here brady cardiac
∴ SA node may also effected
- As it's IWMI + PWMi + RVMi Level of
occlusion very proximal

NOTE:

To find RVMi

(i) ST Elevation in V_3r, V_4r, V_5r, V_6r

(ii) In 12 lead ECG

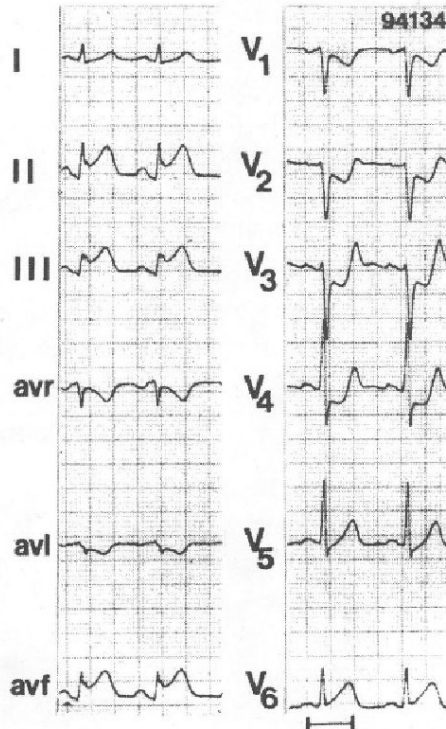
 V_1 will show Discordance with V_2 Usually V_1 minimal ST depression / flat(∴ injury vector will pull up the Right side
lead V_1 ∴ minimal depression)

NOTE:

To find out PWMi

(i) On V_7, V_8, V_9 (ii) On 12 lead ECG look for V_2 will show ST depression &
Reciprocal change of ST Elevation

CIRCUMFLEX CORONARY ARTERY



- ST Elevation : Lead II, III & aVF

∴ IWM

- ST Elevation in lead II & III

∴ LCX effected

- ∴ check for a) PWM
V₂ show ST depression

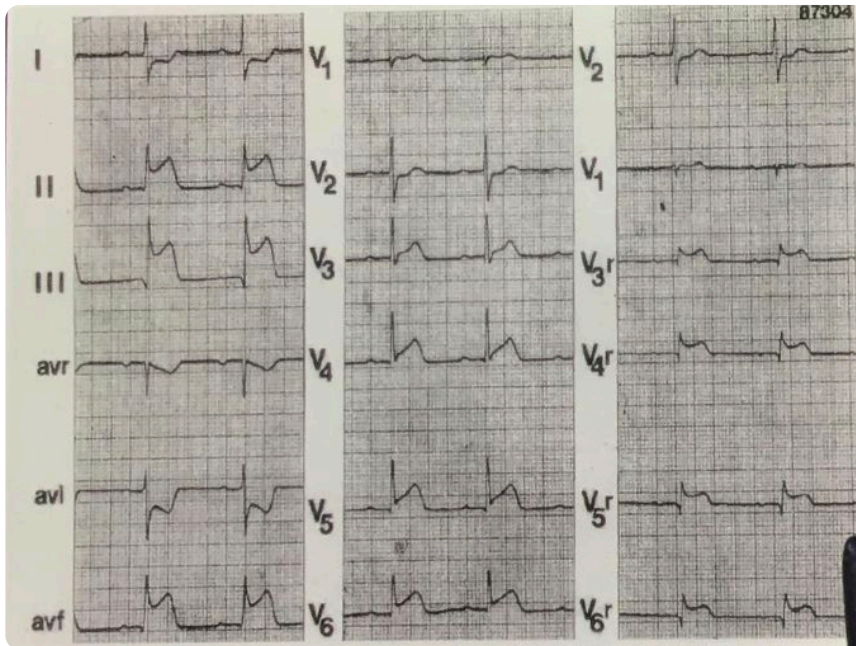
∴ it's PWM also

b) LWM

Check for V₅, V₆ ST elevation

∴ Here LWM also

∴ IWM + PWM + LWM



- STELEVATION IN II, III, aVF
- RECIPROCAL CHANGES in I & aVL
- STELEVATION III > II

∴ IWM with RCA occlusion

- V₁ showing ST Elevation usually it should be depressed.

∴ RWM

(Also STElevation in V_{3r}, V_{4r}, V_{5r}, V_{6r})

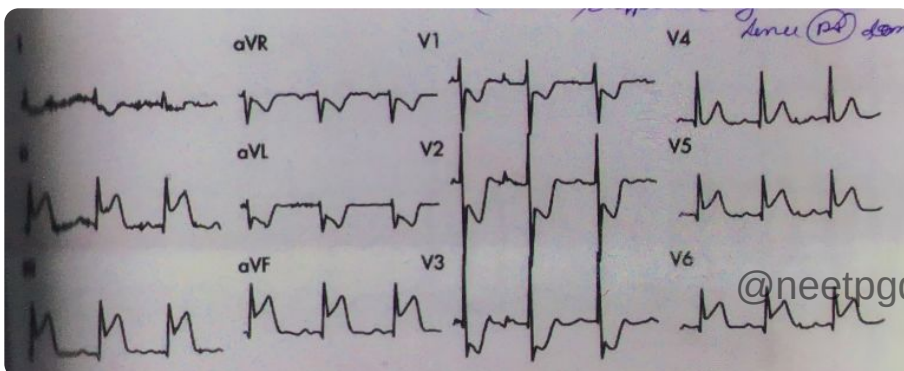
- V₂ showing STElevation.

∴ Posterior wall is spared

- ∴ IWM + RWM

But here V₅, V₆ show ST elevation
The RCA in this case supply the lateral wall also. This is called

WRAP AROUND RCA



- Lead III > II : RCA

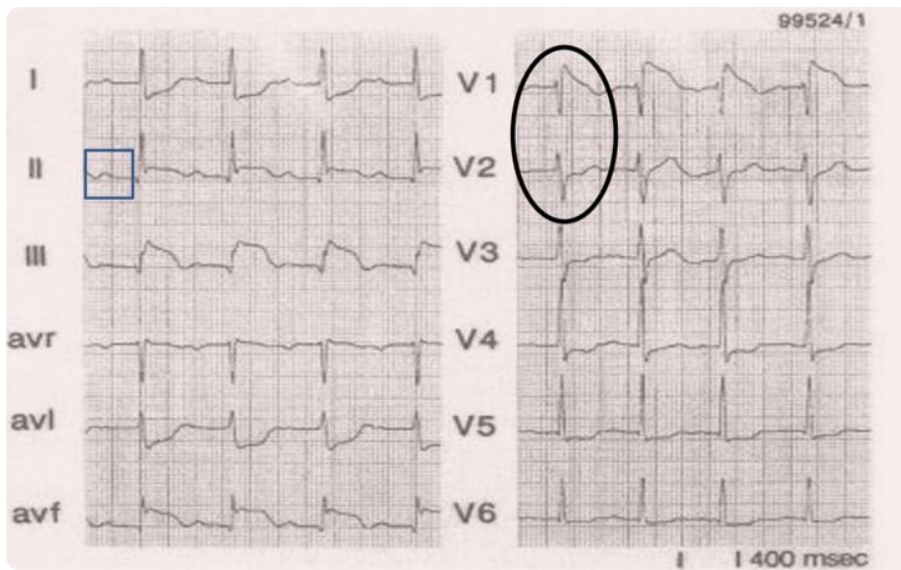
(∴ aVL show max depression)

- V₁-V₂ discordance : ∴ RWM

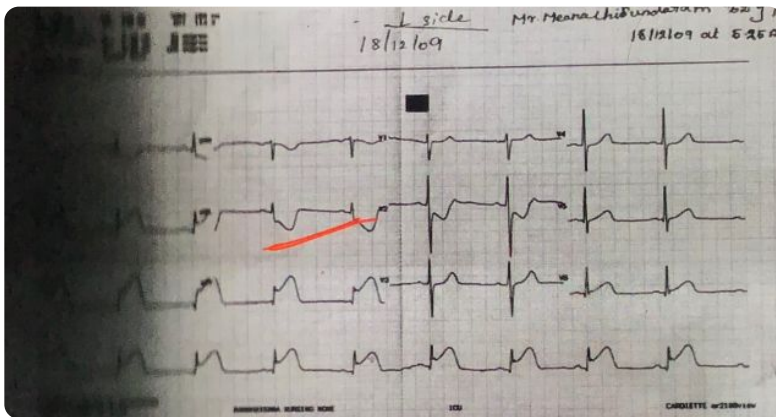
- V₂ ST depression : ∴ PWM

- V₅ & V₆ STElevation : ∴ Lateral wall is also supplied

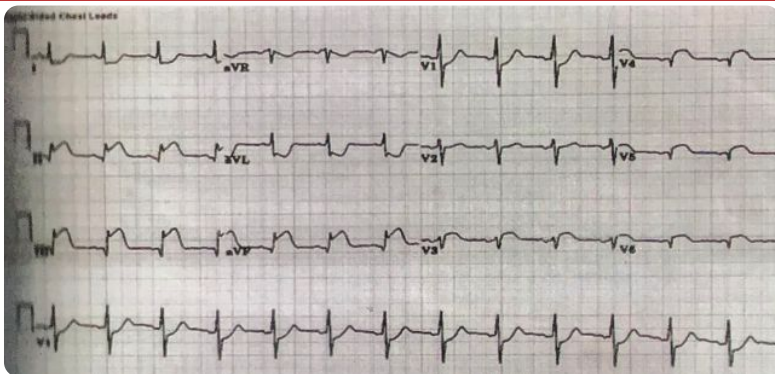
∴ Wrap around RCA



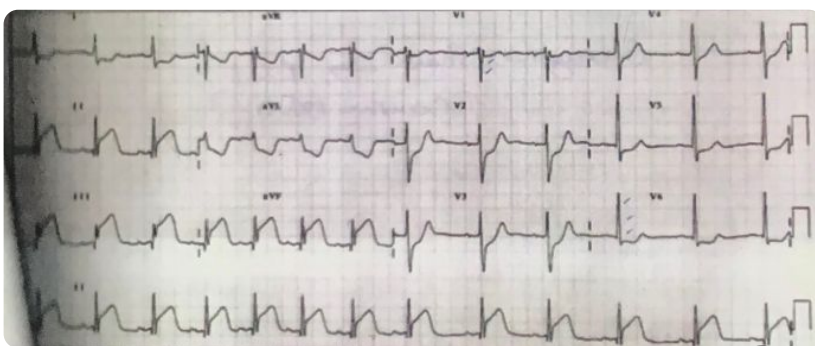
- Lead III > II : $\therefore$ Lcx
- Reciprocal changes in aVL
- V₁ showing ST elevation
 $\rightarrow \therefore$ RVMI
- Lead II, III, aVF ST elevation
 $\rightarrow$ IWMI
- No depression of ST : $\therefore$ No PWMI
- Rate $\approx$ 100 beats/min.
- $\therefore$ RVMI + IWMI



- Lead III > II $\rightarrow \therefore$ RCA
- V₁ discordance with V₂
 $\rightarrow \therefore$ RVMI
- V₂ depression of ST
 $\rightarrow \therefore$ PWMI
- $\therefore$ it's IWMI + RVMI + PWMI proximal occlusion

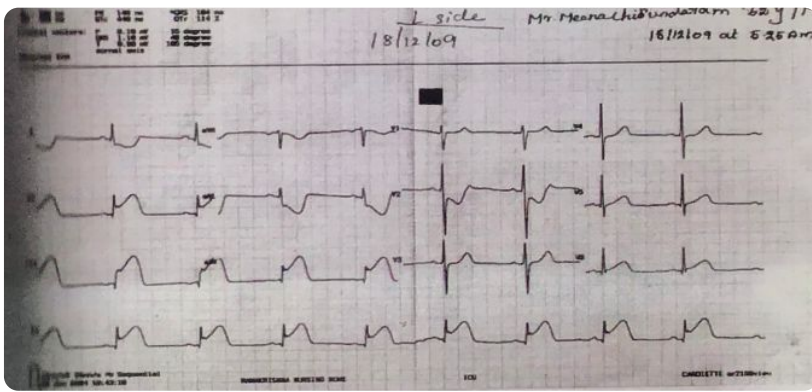


- Lead III > II : RCA Occlusion
- No depression in V₂
 $\rightarrow$ No PWMI
- V₁ & V₂ showing some discordance
 $\rightarrow$ No confirmation
- $\therefore$ only IWMI / Distal RCA

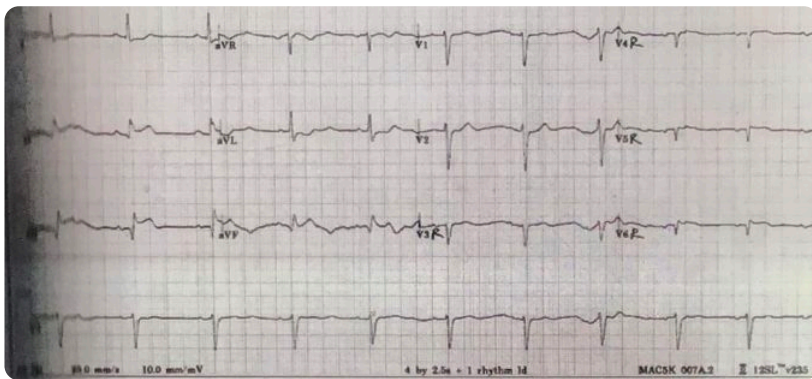


- Lead III > Lead II : RCA
- Discordance of V₁ & V₂
- V₂ ST depression : $\therefore$ PWMI
- $\therefore$ IWMI + RVMI + PWMI

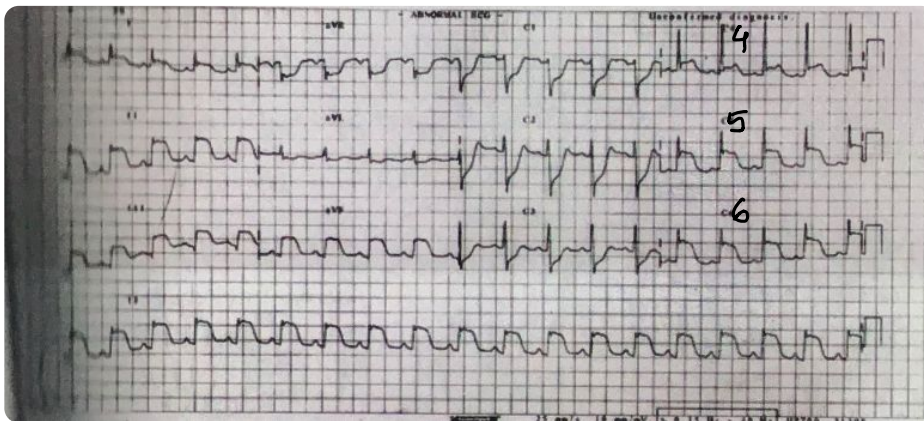
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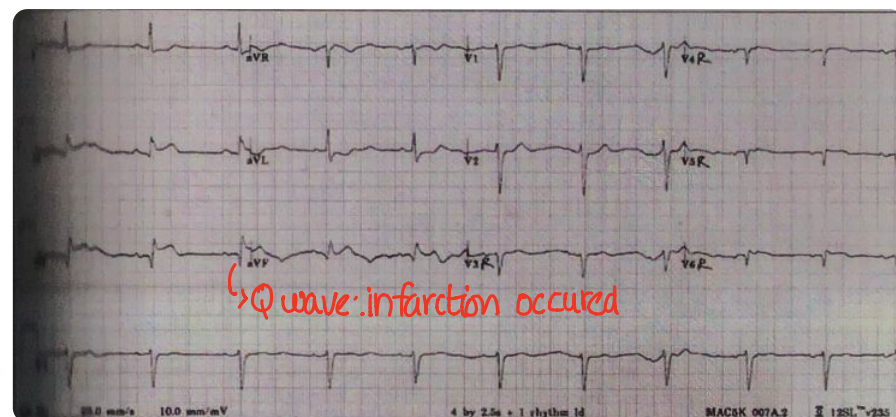
- Lead III > Lead II : RCA
- V₁-V₂ discordance
- V₂ depressed
- ∴ IWM I + PWM I + RVMI



- Lead III > II : ∴ RCA
- V₁ ST flat :
V_{4R}, V_{5R}, V_{6R} : Mild degree of ST elevation (RVMI)
- V₂ ST no depression : ∴ No PWM I
- ∴ IWM I + RVMI
(∴ Proximal RCA occlu.)

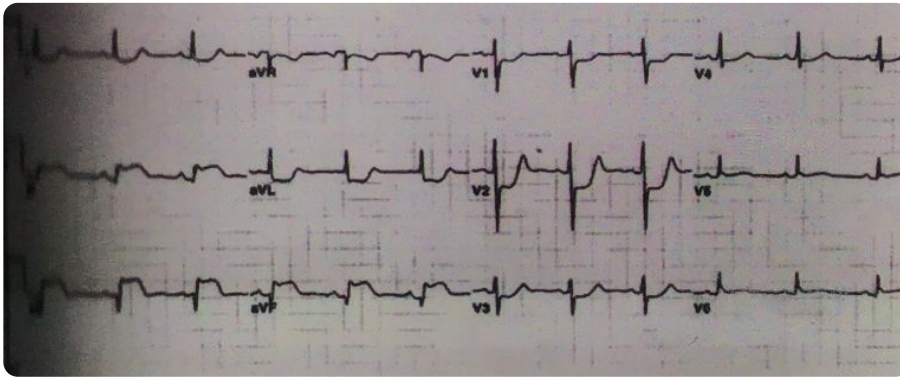


- Lead II > III : LCX
- V₅, V₆ ST elevation
- V₂ ST depression
- ∴ PWM I + LWMI + IWM I

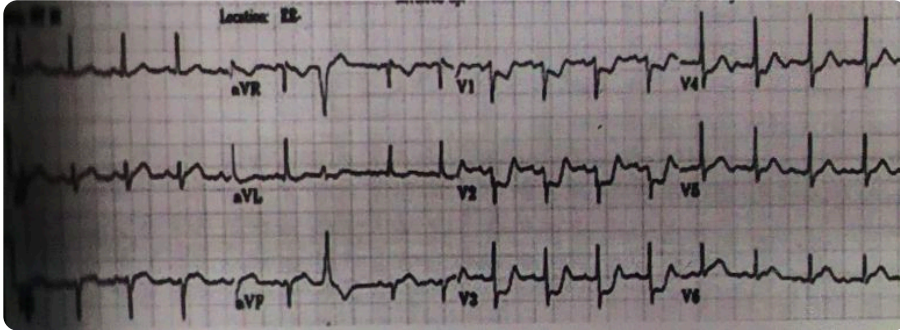


- Lead III > II with a Q wave
↳ ∴ RCA
- No ST depression : No PWM I on V₂
- V₁ flat :
V_{4R}, V_{5R}, V_{6R} : ST elevation
- ∴ RVMI
- ∴ Proximal RCA

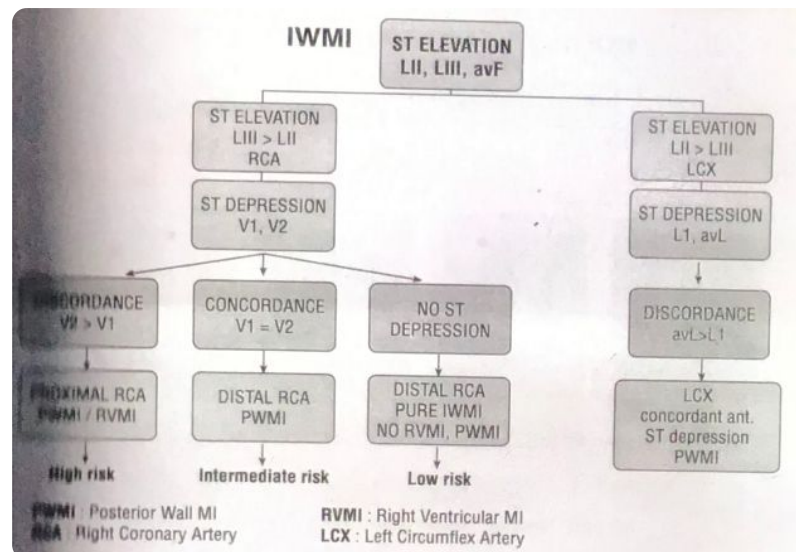
@notpgdiscussion



- Lead III > II : RCA
- V₁-V₂ Discordance
 ↳ ∴ RVMI
- V₂ depression : ∴ PWMI
- ∴ IWMI + PWMI + RVMI



- ST depression in V₁, V₂, V₃
 2 possibilities
 ↳ Antero Septal NSTEMI ↳ PWMI
- Profound depression in V₂
 Chance of PWMI is high
 (V₁ depression < V₂)
 ∴ Do a **BEDSIDE ECHO**



Summary

IWMI → Reciprocal changes in I, aVL (or) V₁-V₆

Lead III > II ST Elevation

RCA

V₁-V₂ Discordance
 with ST depression
 RVMI(+) PWMI(+)

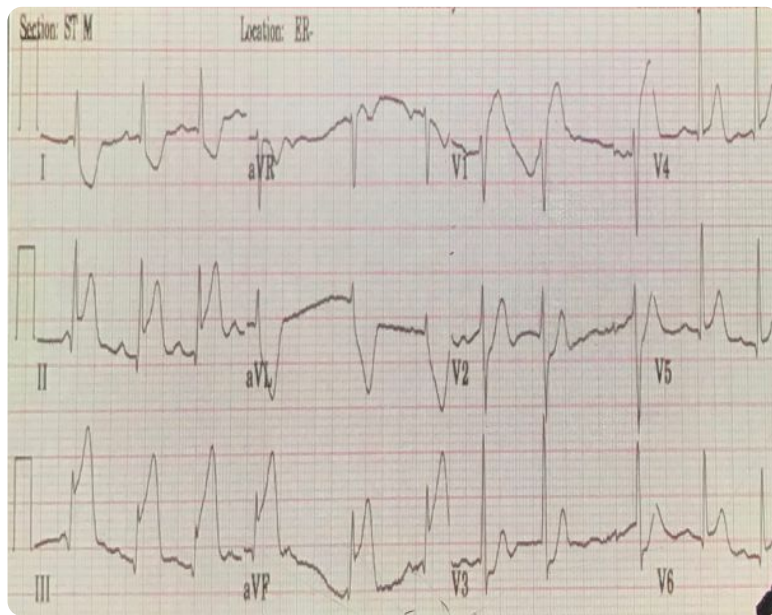
V₁, V₂ Depression
 with no discordance
 PWMI(+)

V₁, V₂ Normal
 IWMI

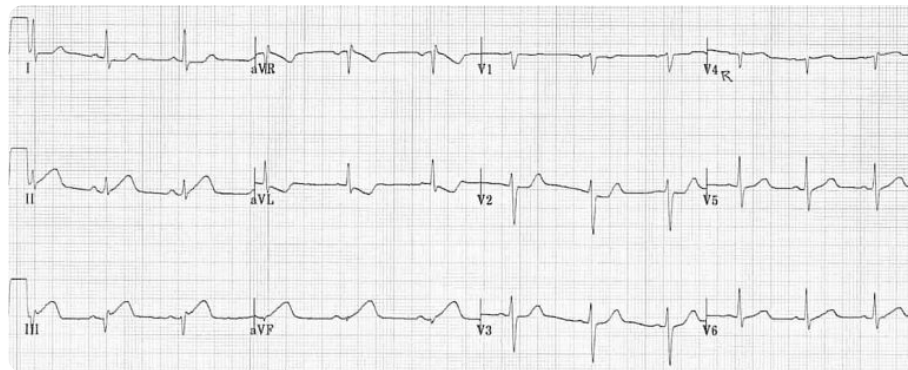
Lead II > III ST Elevation

LCX

LWMI : ST elevation V₅, V₆
 PWMI : ST depression in V₁, V₂, V₃

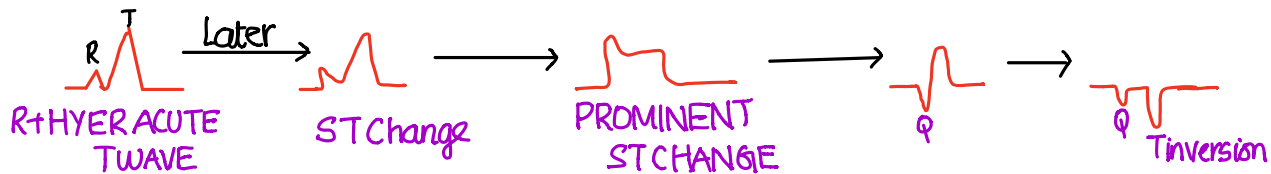


- Lead III > II : RCA
- V₁ showing ST elevation : ∴ RVMI
- Some depression in V₂ : ∴ PWMI
- But here
PR interval are not regular
PP interval are regular
↓
3° Heart block
- IWM + Complete Heart block
Pt require Pacing



- LEAD III > II
- V₁-V₂ DISCORDANCE
- V_{4R} : ST elevation
- ∴ RVMI + IWM

PROGRESSION OF MI



Post Myocardial Infarction

Hyperacute phase



Fully evolved phase

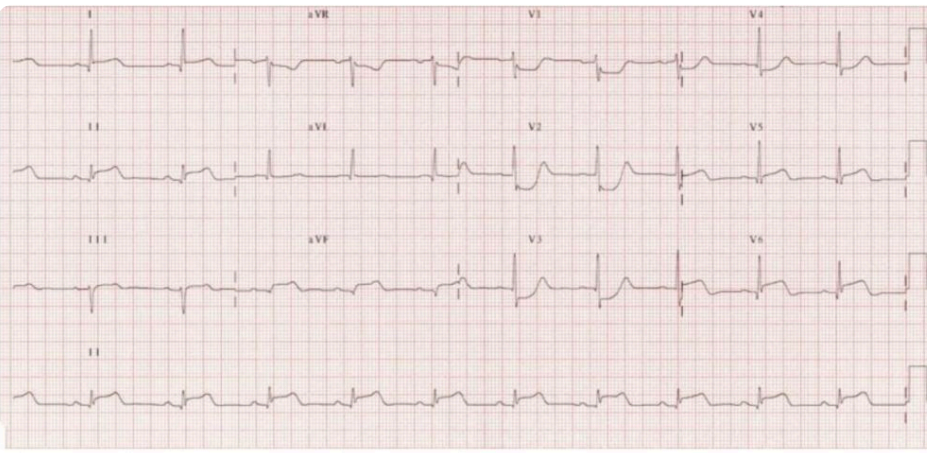


Resolution phase

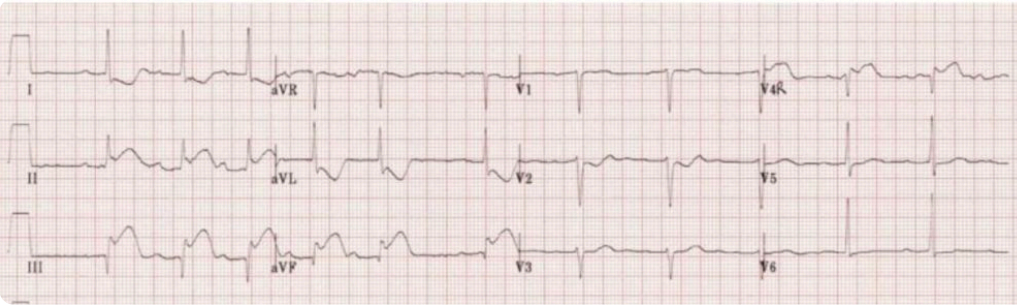


Stabilized chronic phase





- LEAD II > III : LCX
- V₂ DEPRESSION OF ST
↓
PWMI
- V₅, V₆ ST Elevation
↓
LWMI



- LEAD III > II : RCA
- V₁-V₂ DISCORDANCE
- RVMI, LWMI, Most often PWMI.

LEFT CORONARY ARTERY

- Originate from Left interventricular sulcus
- First Branch : **DIAGONAL BRANCH (D₁)** : Parallel TO CIRCUMFLEX A.
 ↳ Supply BASAL ANTEROLATERAL WALL
 is also called as High Lateral
 ↳ Leads I, aVL
- **SEPTAL BRANCH (S₁)** : Supply
 ANTERIOR 2/3rd of INTER VENTRICULAR SEPTUM
 CONDUCTING SYSTEM DISTAL TO BUNDLE of HS
 Leads V₁-V₂
- **DIAGONAL BRANCH (D₂)** : Supply
 ANTERIOR WALL + APEX
 Leads V₂-V₄
- **LEADS V₅-V₆** : BY LCX or LAD

Occlusion Proximal LAD
↓
involve V₁-V₆, I, aVL
EXTENSIVE ANTERO-LATERAL MI

Occlusion after origin of LCX
V₁-V₄, I, aVL
ANTEROSEPTAL MI
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V₁-V₆ involvement
ANTEROLATERAL MI
V₁-V₂ involvement
SEPTAL MI

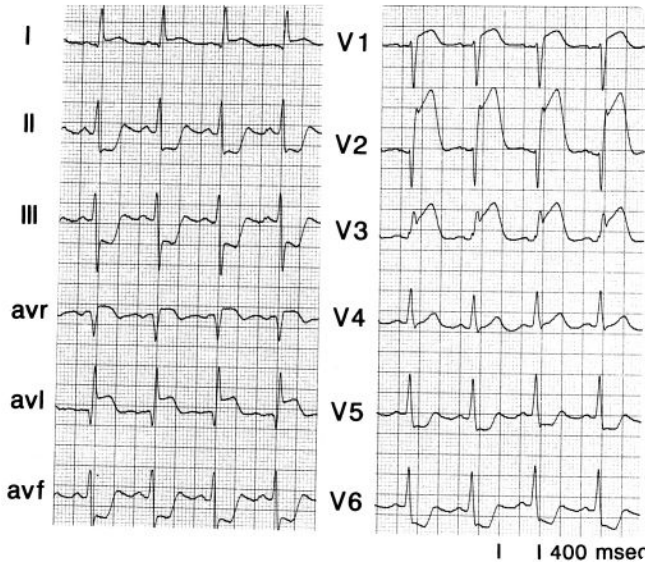
✓ Anterior wall MI is more severe as it can cause

- (i) TACHYARRHYTHMIA
- (ii) RBBB > LBBB
- (iii) CARDIOGENIC SHOCK

✓ SITE OF LESION

- (i) PROXIMAL (before D_1 & S_1)
- (ii) Between D_1 & S_1
- (iii) DISTAL TO S_1

- EXTENSIVE ANTEROLATERAL MI
- ANTEROSEPTAL MI
- PURE ANTERIOR WALL MI



• RECIPROCAL CHANGES ARE SEEN

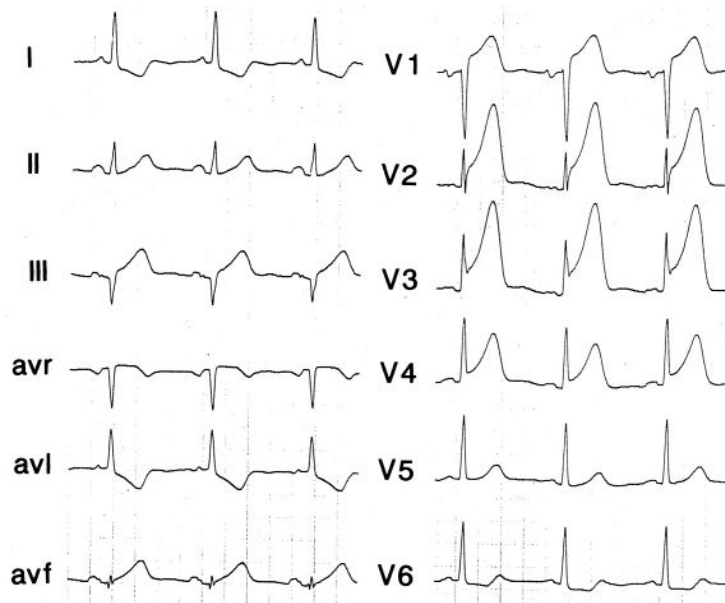
• STELEVATIONS : V_1, V_2, V_3, I, aVL

∴ Diagonal br involved ← Significant
SITE OF LESION
ABOVE THE ORIGIN OF
DIAGONAL BRANCH
• V_1, V_2 involved
∴ SEPTUM involved

• V_2, V_3 ST ELEVATION : ANTERIOR WALL

• ∴ LAD OCCLUSION PROXIMAL TO D_1

• NO ST ELEVATION ON V_5, V_6 ∴ LCX Supply



• RECIPROCAL CHANGES ARE SEEN

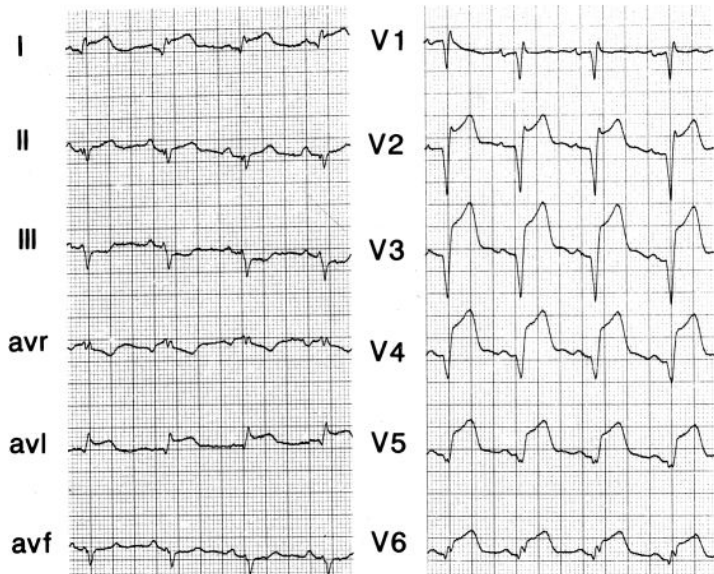
• $V_1 - V_4$ STELEVATION:
ANTEROSEPTAL

• I & aVL : NO ST ELEVATION
∴ D_1 SPARED

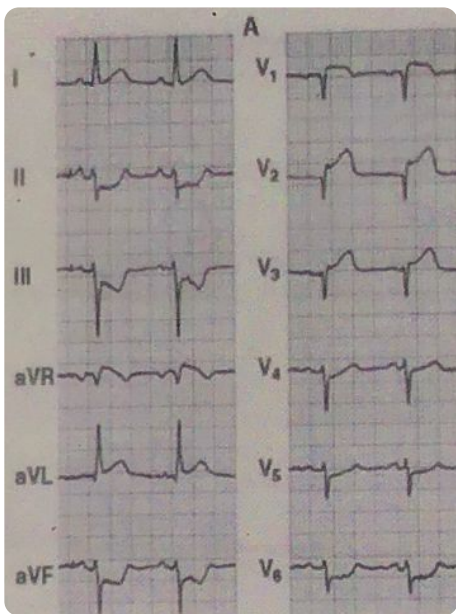
• V_5 & V_6 : NO STELEVATION
∴ CIRCUMFLEX SPARED

• V_1 & V_2 : STELEVATION
∴ BEFORE SEPTAL ARTERY

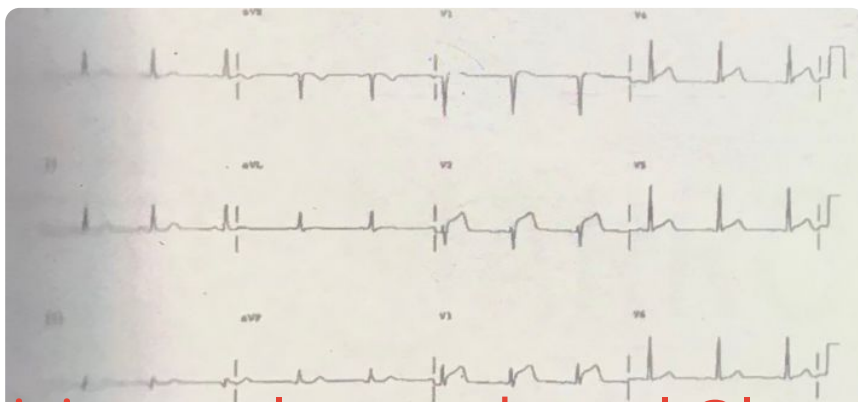
• ∴ SITE OF OCCLUSION:
BETWEEN D_1 & S_1



- RECIPROCAL CHANGES ARE SEEN
 - I, aVL : ST ELEVATION
: $\therefore$ LESION PROXIMAL TO FIRST
DIAGONAL (or)
LCX OCCLUDED
 - V₅-V₆ : ST ELEVATION
 - V₁-V₆ : ST ELEVATION
- ↓
- $\therefore$ VERY PROXIMAL LCA OCCLUSION



- RECIPROCAL CHANGES ARE SEEN
 - I, aVL : ST ELEVATION
 - V₁-V₂ : ST ELEVATION
 - V₅-V₆ : NO ST ELEVATION (LCX SPARED)
- ↓
- $\therefore$ PROXIMAL OCCLUSION BEFORE D₁

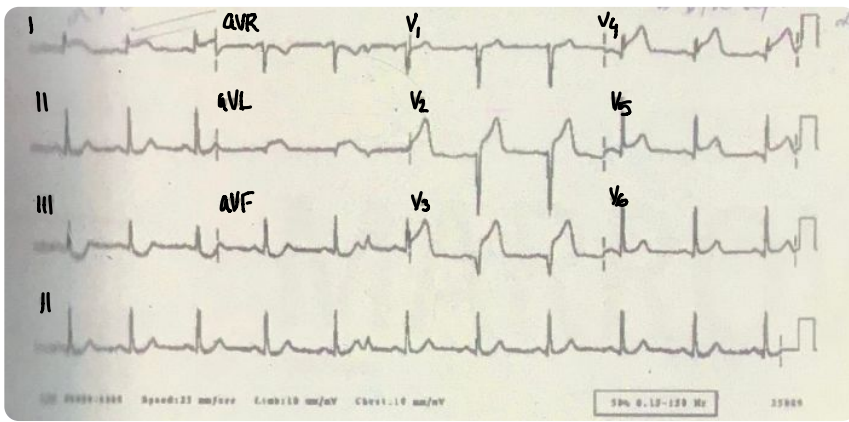


- NO RECIPROCAL CHANGES
- ↓
- $\therefore$ NOT MI

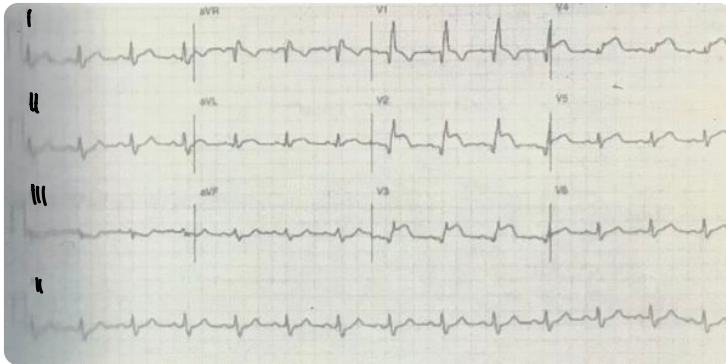
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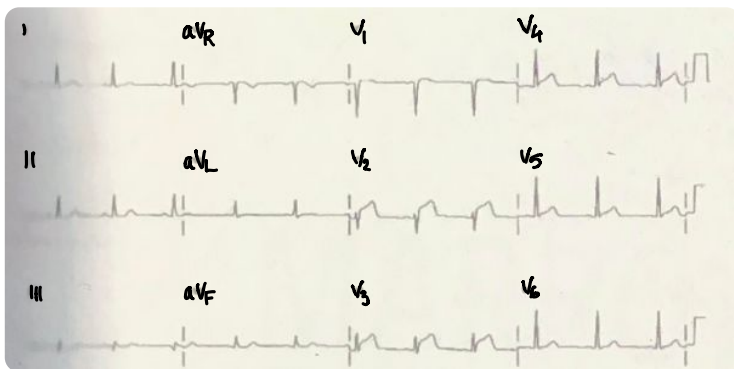
@neetpgdiscussion



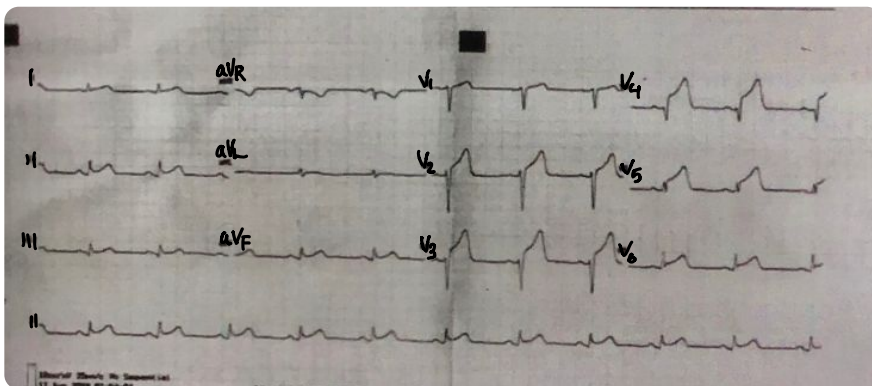
- RECIPROCAL CHANGES ARE SEEN (III & aVF)
- I & aVL : STELEVATION
- V₁-V₅ : STELEVATION
V₆? : ANTEROLATERAL INVOL.
- LESION: PROXIMAL LAD OCCLUSION ABOVE D₁



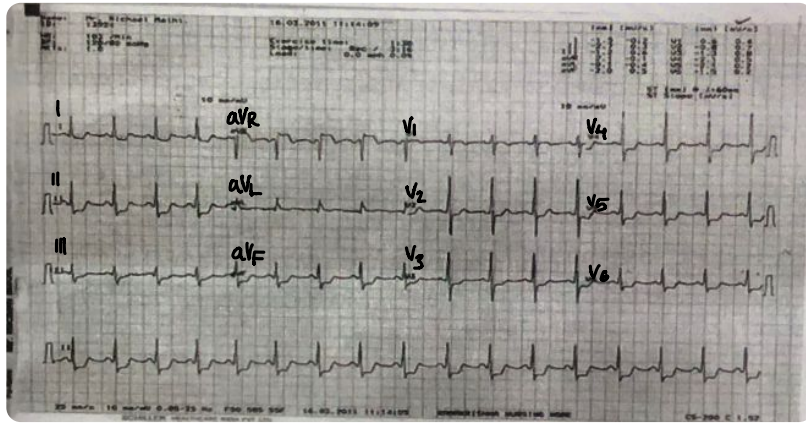
- RECIPROCAL CHANGES ARE SEEN
- I & aVL : STELEVATION
- V₁-V₆ : STELEVATION
- LESION: PROXIMAL LAD OCCLUSION ABOVE D₁



- NO RECIPROCAL CHANGES
↓
∴ NOT MI

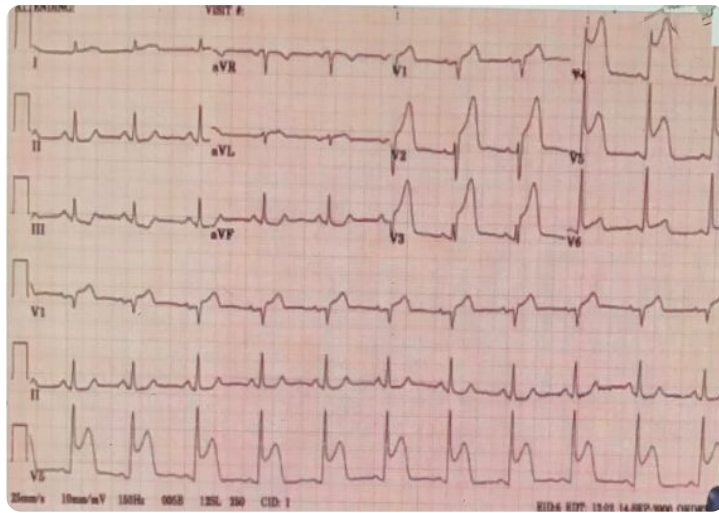


- RECIPROCAL CHANGES ARE SEEN
- V₁-V₆ : STELEVATION
- II - III : STELEVATION
→ After supplying Ant. & Lat. wall a small wrap supply INFERIOR WALL ALSO WRAP AROUND LCA

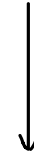


OSTIAL LAD OCCLUSION/LEFT MAIN OCCLUSION

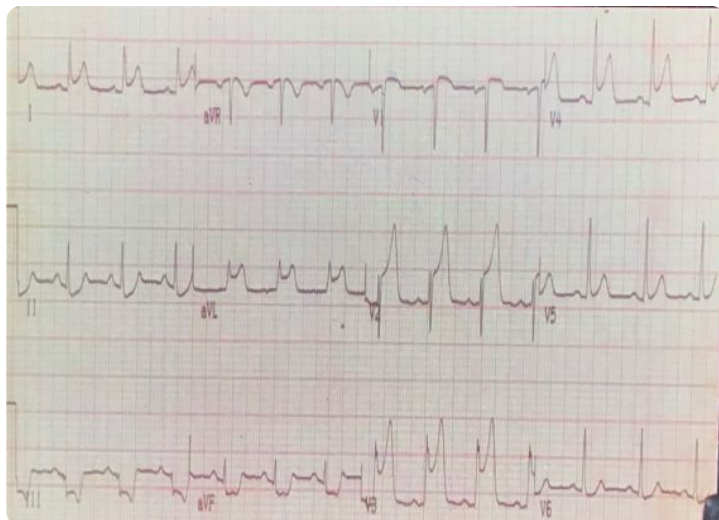
- ST ELEVATION IN aVR
- NO ROLE OF THROMBOLYSIS
IMMEDIATELY SHIFT TO CATH LAB



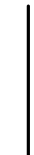
- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION: V₁-V₆, I, aVL



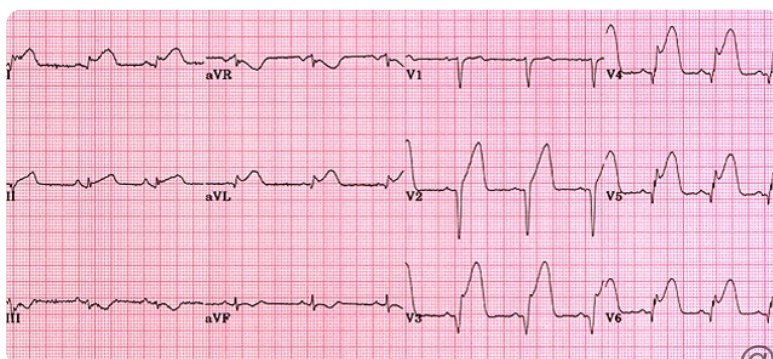
PROXIMAL OCCLUSION ABOVE
THE ORIGIN OF LCX



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION: V₁-V₄, I, aVL



LAD above origin of D₁

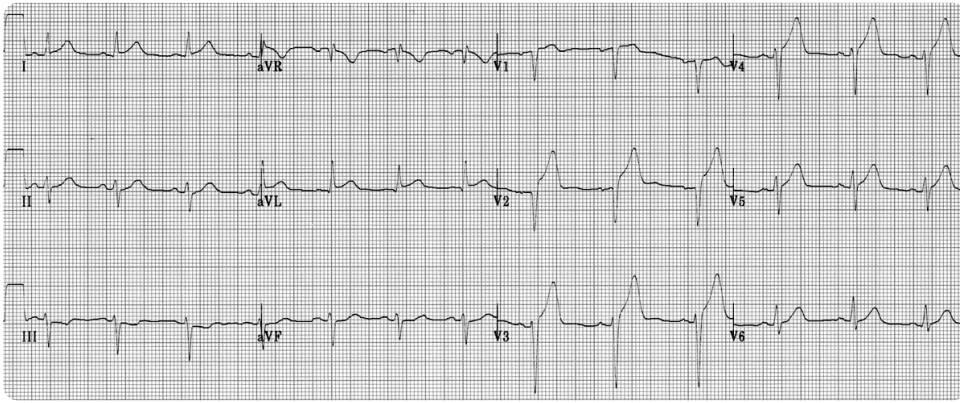


- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION: V₂-V₆, I, aVL



PROXIMAL OCCLUSION ABOVE
THE ORIGIN OF LCX

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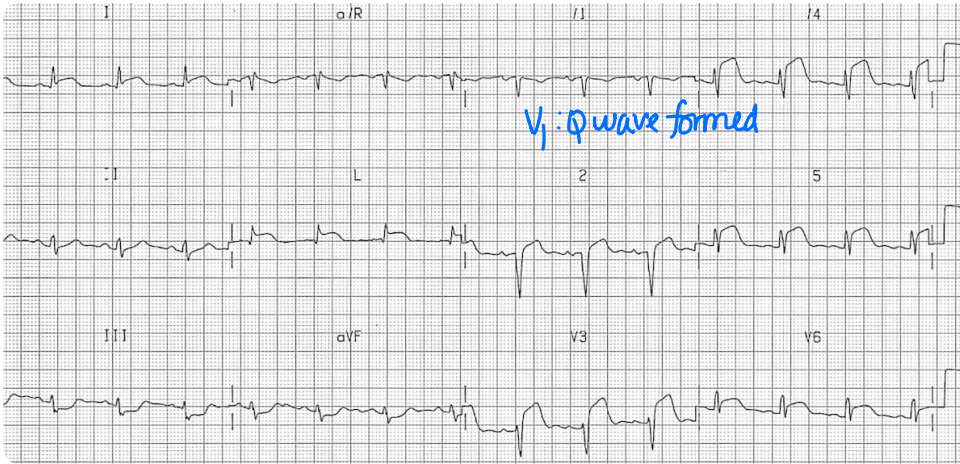


• RECIPROCAL CHANGES ARE SEEN

• ST ELEVATION:
V₁-V₄, I, aVL



LAD above origin of D₁

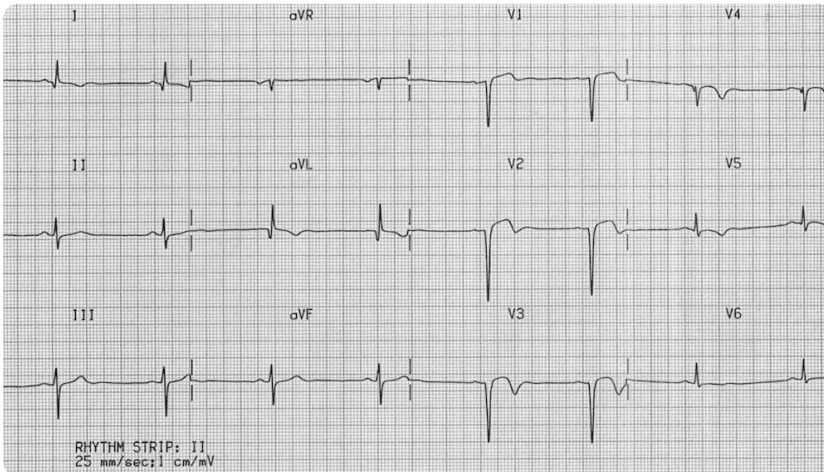


• RECIPROCAL CHANGES ARE SEEN

• ST ELEVATION:
V₁-V₆, I, aVL



PROXIMAL OCCLUSION
ABOVE THE ORIGIN
OF LCX



• RECIPROCAL CHANGES ARE SEEN

• NO ACTIVE ACUTE RECIPROCAL CHANGES

• QS Complex in V₁-V₂

• RESIDUAL ST ELEVATION IN V₁-V₃: **LEFT VENTRICULAR ANEURYSM MORPHOLOGY**

• OLD MI WITH PERSISTENT ST ELEVATION

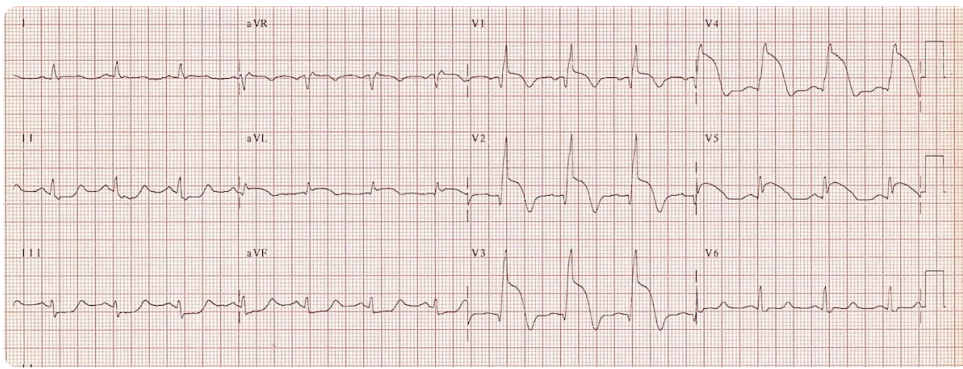


• ST ELEVATION IN aVR



OSTIAL LAD OCCLUSION

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• RECIPROCAL CHANGES ARE SEEN

• ST ELEVATION:

V_1-V_6 , I, aVL



PROXIMAL OCCLUSION
ABOVE THE ORIGIN
OF LCX

SUMMARY

aVR : ST ELEVATION → Proximal ostial stenosis

I, aVL, V5, V6 ST ELEVATION : ABOVE CIRCUMFLEX

I, aVL ST ELEVATION : Proximal to D₁

V₁-V₄ STELEVATION : B/W D₁ & S₁ → ANTEROSEPTAL

V₂-V₄ ST ELEVATION : AFTER S₁ → PURE ANTERIOR

SIGNIFICANT ST ELEVATION

CONVEX UPWARD elevation is taken as Significant ST elevation.

NON CONCAVE
(CONVEX) ✓



CONCAVE
(SADDLE SHAPED)



Learning Bite

A convex ST segment shape is more likely to be associated with AMI than a concave shape

Pitfall

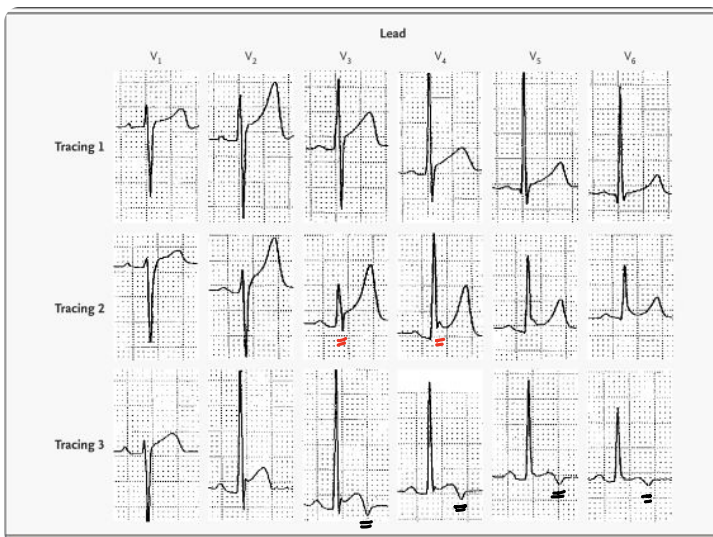
However, do not assume that because ST segment elevation is not convex that it cannot be a STEMI @neetpgdiscussion

DIFFERENTIAL DIAGNOSIS OF ST ELEVATION



- Here
- * LOW VOLTAGE COMPLEXES
 - * GLOBAL ST ELEVATION
 - * NO RECIPROCAL CHANGES
 - * CONCAVE UPWARDS
 - * PR SEGMENT DEPRESSION

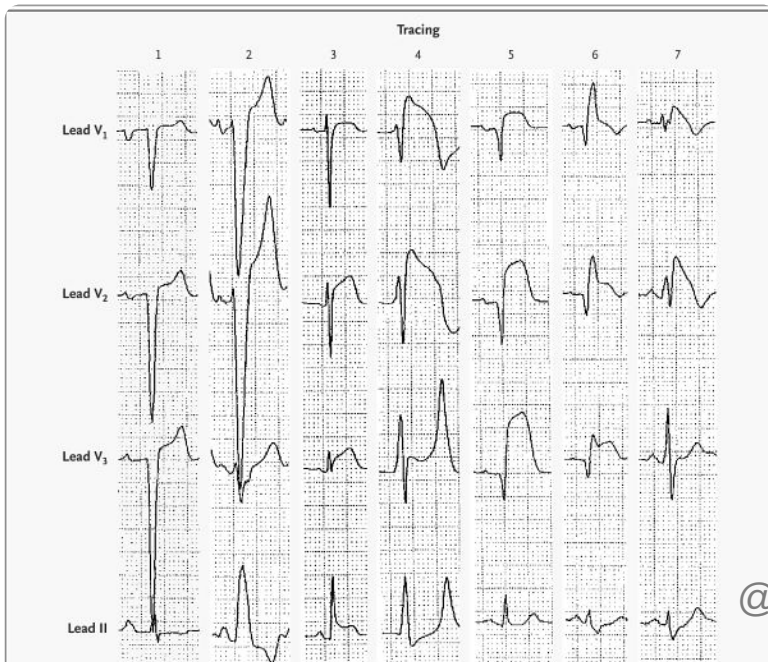
PERICARDITIS



Tracing I: CONCAVE UPWARD ST ELE
NO RECIPROCAL CHANGES
It's NORMAL ECG

Tracing I: CONCAVE UPWARD ST ELE
NOTCH AT "J" POINT
EARLY REPOLARISATION SYN.

Tracing III: ASSYMETRICAL T-INVERSION
NORMAL VARIANT



Tracing 5: TYPICAL MI PATTERN

Tracing 6: TYPICAL MI PATTERN

Tracing 7: BRUGADA SYNDROME
Pattern in V₁ & V₂

Tracing 4: HYPER KALEMIA
Tall peaked T wave in V₃

Tracing 3: PERICARDITIS

Tracing 2: LBBB PATTERN

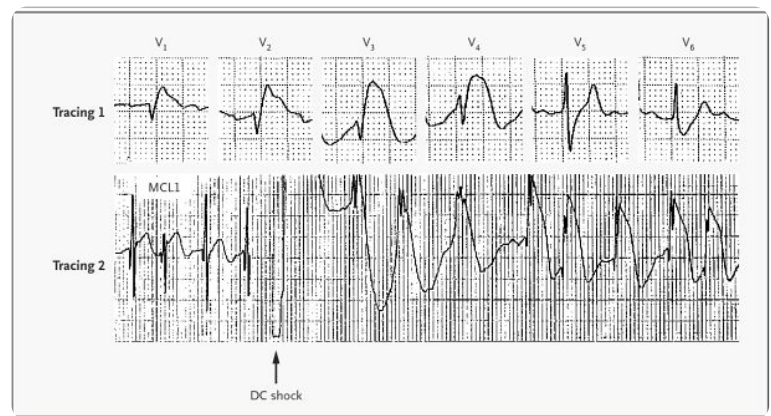
Tracing 1: LVH (Left Ventricular Hypertrophy)

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More than just notes...

Conditions.	
Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V ₂ Concave
Early repolarization	Most marked in V ₄ , with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V ₃ through V ₅ with inverted T waves Short QT, high QRS voltage
Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome	rSR' in V ₁ and V ₂ ST-segment elevation in V ₁ and V ₂ , typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and antero-septal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III



Tracing I : **PULMONARY EMBOLISM**
in V₃ & V₄

Tracing II : **PATTERN AFTER DC SHOCK**

CAUSES OF ST ELEVATION

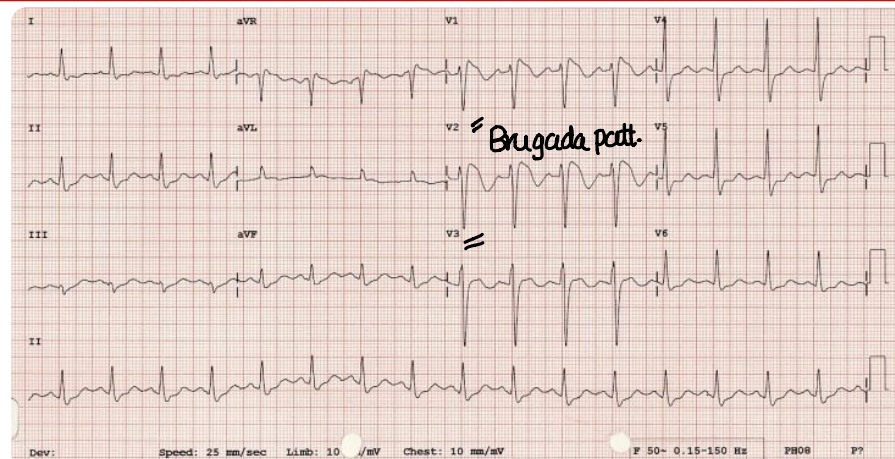
(i) ACUTE M

(ii) PRINMETALS ANGINA

(iii) VENTRICULAR ANEURYSM

(iv) ACUTE PERICARDITIS

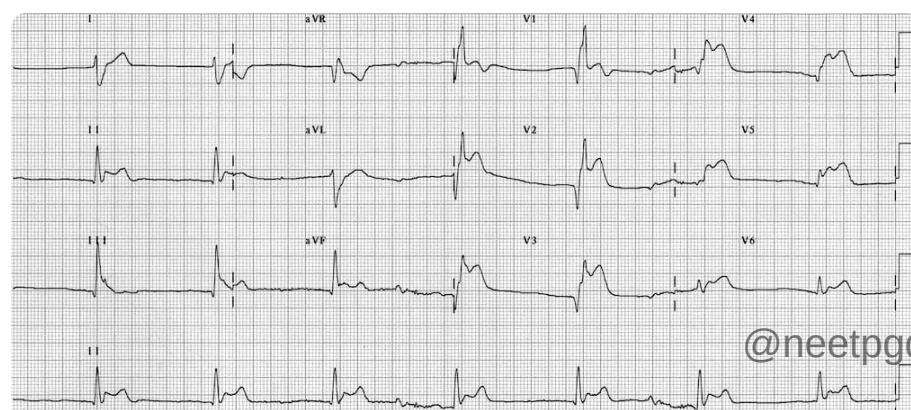
(v) ERS PATTERN



• NO RECIPROCAL CHANGES

• **CHANGES ONLY IN V₁ & V₂**

↓
BRUGADA PATTERN
(prone to sudden cardiac death)



• NO RECIPROCAL CHANGES

• Typical convex upward elevation in all leads

• Pt PRESENT WITH CHEST PAIN

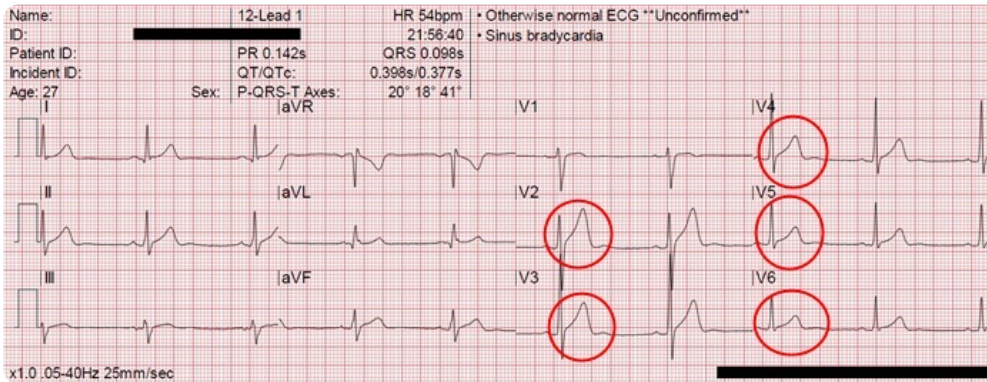
↓
On Angio : **NORMAL**
called as **TAKOTSUBO CARDIO MYO-PATHY / STRESS CARDIOMYOPATHY / LV APICAL BALLOONING SYNDROME**

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TAKOTSUBOCARDIO MYOPATHY :

- * Benign condition
- * Recovers
- * normal angiogram

There is HYOKINESIA of APEX
+
HYPER CONTRACTILE BASE
↓
APICAL BALLOONING OF LV



EARLY REPOLARISATION SYNDROME PATTERN

- * Slight elevation in J point (or) knotch in J point.

UNIQUE COMPLICATIONS IN MI

1. LV FREE WALL RUPTURE

- seen in ANTERIOR WALL MI (MC)
- can occur at 1-7 days (Generally 3-5 days)
- CARDINAL SIGN : CHEST PAIN
CARDIAC TEMPONADE
JVP ELEVATION
PULSUS PARADOXUS
ELECTRO MECHANICAL DISSOCIATION
HYPOTENSION
CARDIOGENIC SHOCK

- CARDIAC TEMPONADE : HYPOTENTION
MUFFLED HEART SOUNDS
JVP ELEVATION

} BECK'S TRIAD

2. PAPILLARY MUSCLE RUPTURE

- Occurs immediately
- Seen in INFERIOR WALL MI (MC)
- Presentation : Pulmonary Edema
- Characterised by (i) ACUTE ONSET MR

NEW SYSTOLIC MURMUR at APEX

(ii) V WAVES ON JVP

- TREATMENT : IABP + Surgical Correction

↓
Intra aortic balloon pump

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3. VENTRICULAR SEPTAL RUPTURE

- Characterised by onset within 3-7 days
- Presentation : CHEST PAIN
SHORTNESS OF BREATH
HYPOTENSION
- Identified by : (i) ACUTE ONSET MR (New Systolic murmur at Parasternal area)
(ii) LOUD S2

ONE LINERS

- Killip Classification is used to classify HF in patient with MI
- JVP Elevation is seen in
RVMI : along with MI
LV free wall Rupture : 3-5 days after MI
- S₃ GALLOP RHYTHM : —————→ SYSTOLIC LV DYSFUNCTION
- S₄ : —————→ DIASTOLIC DYSFUNCTION
- MR is complication of (i) Papillary Muscle rupture
(ii) Ventricular septal rupture (3 days after MI)
- MRI : MOST SENSITIVE TEST TO DETECT INFARCTION
- CALCIUM CHANNEL BLOCKERS NO ROLE IN ACUTE MI

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HYPERTROPHY & BUNDLE BRANCH BLOCK

SOKOLOW LYON INDEX

- CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY (LVH)

$$V_1 \text{ or } V_2s + V_5 \text{ or } V_6R > 35 \text{ mm}$$

(OR)

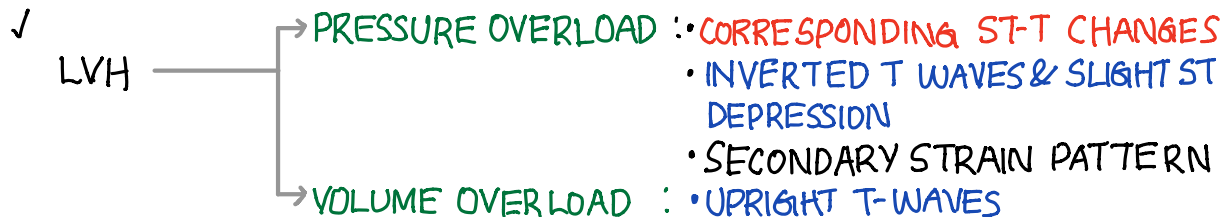
$$R \text{ WAVE IN } aVL > 11 \text{ mm}$$

NOTE :

✓ LVH + Left Axis Deviation (LAD)

(i) In an old patient → Chronically Hypertensive patient

(ii) In a 30 yr old → Hypertensive End-organ damage out of proportion to degree of BP



✓ IF LVH + Strain + LAD LOOK FOR LBBB



LVH WITH STRAIN V/S MI ST-T CHANGE

* Asymmetrical T wave inversion



* ST which does not dip so much from baseline

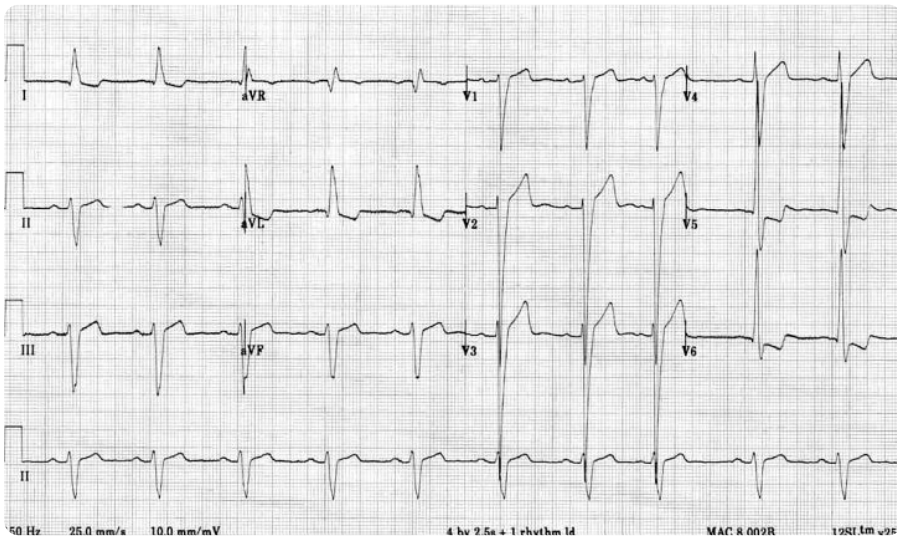
* Symmetrical T wave inversion



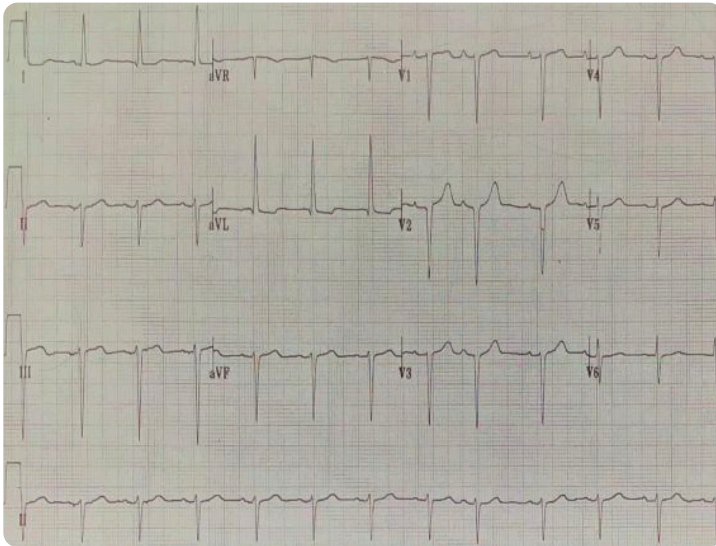
* Severe dip from baseline



LV strain-pattern: ST depression and T wave inversion in the lateral leads



- SLIGHTLY WIDE QRS COMPLEX
- AXIS OF ECG : B/W I & aVL
∴ **LEFTWARD AXIS**
- $V_{1S} + V_{5R} > 35\text{mm}$
∴ **LVH**
- INVERTED T WAVES & SLIGHT ST DEPRESSION ∴ **PRESSURE OVERLOAD**
- Slightly wide QRS COMPLEX in I, aVL, $V_5 - V_6$ ∴ **LBBB MORPHOLOGY**



- NARROW QRS COMPLEX
- LEFT AXIS DEVIATION
- $V_{1S} + V_{5R} < 35\text{mm}$
But a $aVL > 11\text{mm}$ ∴ **LVH**
- But there is NO LBBB Morphology

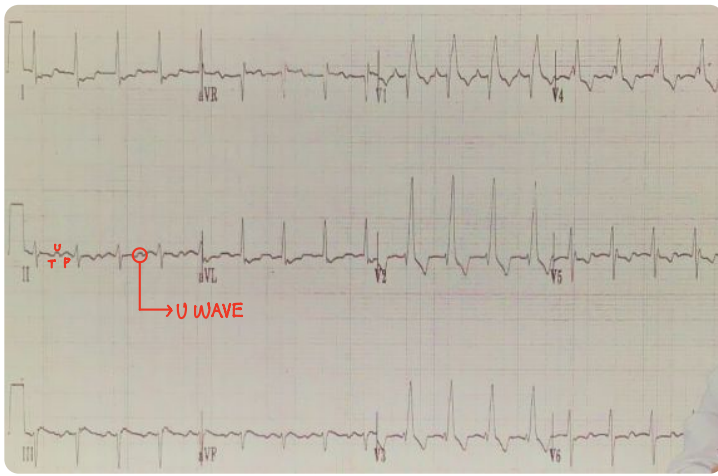
RIGHT VENTRICULAR HYPERTROPHY

- CRITERIA : TALL R WAVE in V_1 i.e. $> 7\text{mm}$ (OR)
 $R/S \text{ RATIO} = > 1$
- RBBB MORPHOLOGY : r-r' or r-s-r' Pattern in $V_1 - V_2$ & M pattern in $V_5 - V_6$

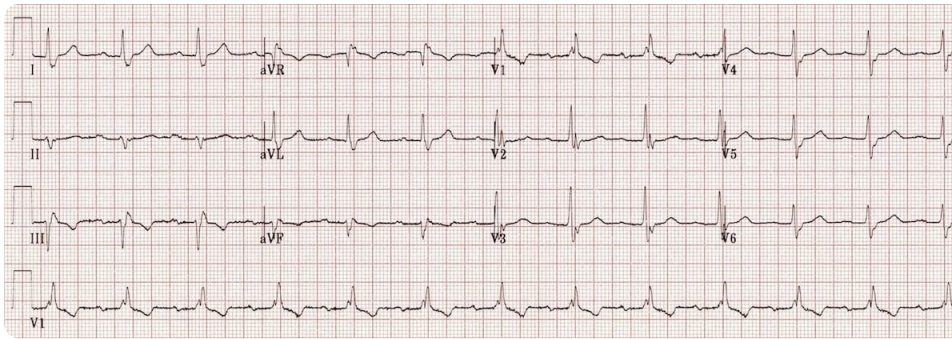
NOTE : Usually LVH is associated with LAD &
RVH is associated with RAD
But if RVH + LAD → **BIFASCICULAR BLOCK**

IF PROLONGATION OF PR INTERVAL
is called **TRIFASCICULAR BLOCK**
i.e. BIFASCICULAR + 1° AV BLOCK
or BIFASCICULAR + 2° AV BLOCK

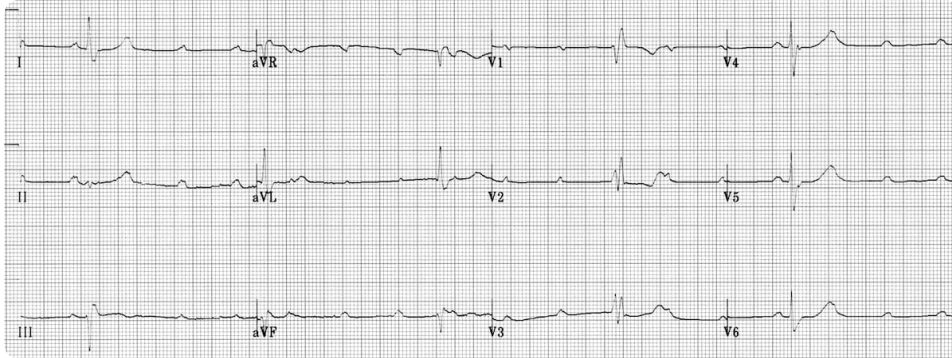
→ RBBB + Left Anterior Fascicle Block
(or)
Left Posterior Fascicle Block



- NARROW QRS COMPLEX
- PR interval : 4 boxes
- U WAVE is considered significant
 - (i) Height of U > $\frac{2}{3}$ rd Height of T wave
 - (ii) U & T are discordance
 → HYPOKALEMIA
- AXIS : LAD
- TALL R WAVE IN V_1 > 7mm
∴ RVH.
- RBBB Morphology : M Pattern in V_1 & V_2
- AS RVH + LAD = BIFASCICULAR BLOCK



- NARROW QRS COMPLEX
- AXIS : LAD
- Typical RBBB Morphology in V_1 - V_2 : BIFASCICULAR BLOCK
- PR PROLONGED
: TRIFASCICULAR BLOCK

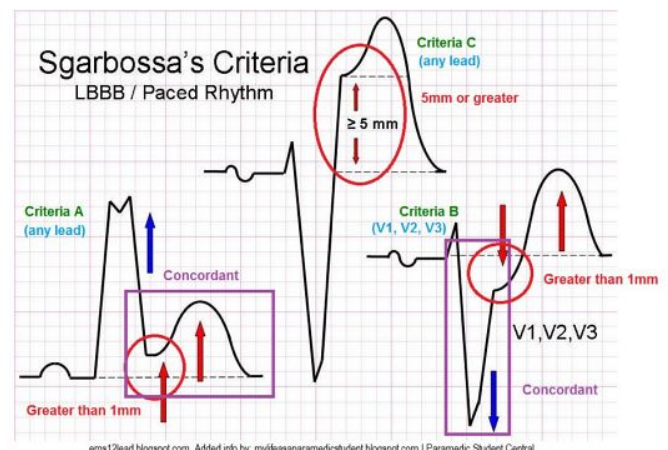


- NARROW QRS COMPLEX
- AXIS : LAD
- Typical RBBB Morphology in V_1 - V_2 : BIFASCICULAR BLOCK
- PR PROLONGED
: TRIFASCICULAR BLOCK

SGARBOSSA'S CRITERIA

- Help in Diagnosis of MI in presence of LBBB

1. ST-segment elevation of 1 mm or more concordant with (in the same direction as) the QRS complex in leads V_5 or V_6
2. ST-segment depression of 1 mm or more concordant with the QRS complex in leads V_1 , V_2 , or V_3
3. ST-segment elevation of 5 mm or more that was discordant with (in opposite direction from) the QRS complex



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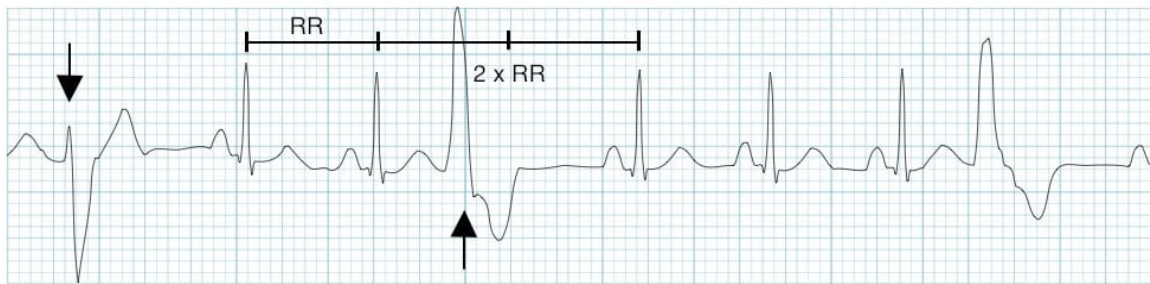


- V₅-V₆ COMPLETE DISCORDANCE
- V₁-V₂ DISCORDANCE
- ST ELEVATION >5mm

↓
LBBB with MI

VENTRICULAR RHYTHM

VENTRICULAR PREMATURE COMPLEX (VPC)/VENTRICULAR EXTRASYSTOLE



- Pt who is in Normal Sinus Rhythm. Then getting VPC. Then again Normal

- * WIDE QRS COMPLEX
- * Always followed by COMPENSATORY PAUSE
i.e the next normal beat arrives after an interval that is equal to double the Preceding R-R interval
- * DISCORDANT ST-T Changes
- * COUPLING INTERVAL: Time taken by PVC to occur from the Preceding QRS Complex

- ↓
- * VPC have CONSTANT COUPLING INTERVAL
 - * If COUPLING INTERVAL is not constant for a VPC

→ Here VENTRICLE MAY TAKE OVER The RHYTHM called as VENTRICULAR PARASYSTOLE

- ↓
- It may form fusion complex
 - Try to take over rhythm

- * INTERPOLATED VPC: VPC BETWEEN TWO SINUS, SUCH IT'S NOT AT ALL ALTERING RHYTHM

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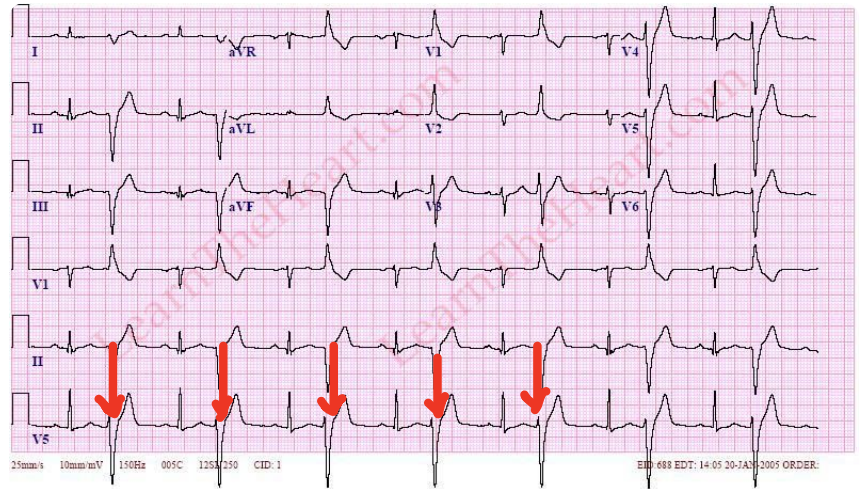
* **R ON T PHENOMENON**: VPC FALL ON THE T WAVE OF PRECEEDING SINUS IMPULSE

VENTRICULAR BIGEMINY

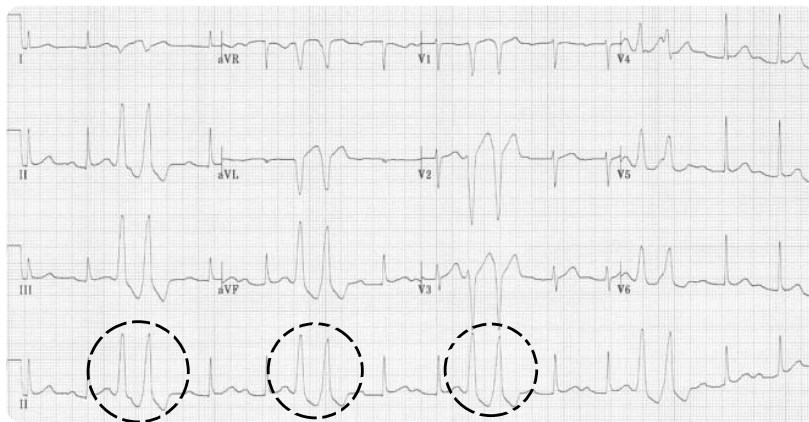
WHEN A VPC ARISES AFTER EVERY SINUS BEAT is called as **VENTRICULAR BIGEMINY** →

WHEN A VPC ARISES AFTER TWO SINUS BEATS called **VENTRICULAR TRIGEMINY**

WHEN TWO VPC'S OCCUR TOGETHER CALLED **COUPLET**



WHEN 3 OR MORE IN A ROW IT'S CALLED **VENTRICULAR TACHYCARDIA**



Couplet

CLINICAL FEATURES - VPC :

- ASYMPTOMATIC
- PALPITATION, MISSED / EXTRA BEAT

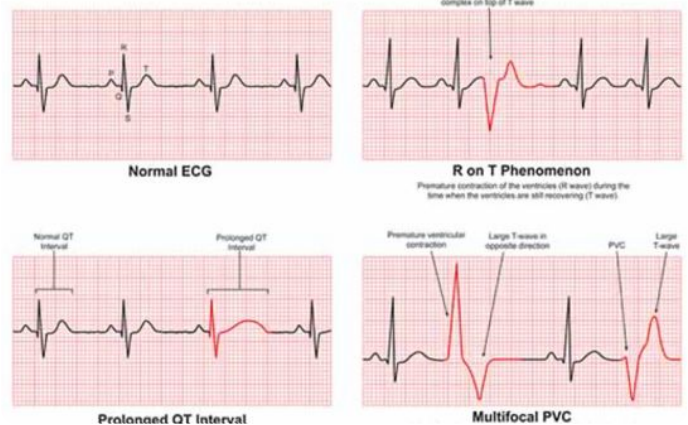
TREATMENT :

- * ANTI-ARRHYTHMIC IN ISOLATED VT - **NO ROLE**
- * NO ROLE FOR PROPHYLAXIS OF VPC
- * GIVE ANTI-ARRHYTHMIC ONLY WHEN VT IS THERE
- * **BETA BLOCKER IN VPCs** IN PATIENT WITH STEMI AND IN PREVENTION OF VF

WHEN VPC'S BECOME DANGEROUS

- * USUALLY BENIGN
- * FREQUENTLY OCCURRING
- * MULTIFOCAL : **VPC WITH DIFFERENT MORPHOLOGY**
- * BIGEMINY
- * FIRST TIME OCCURRING >40 YRS
- * VPC WHICH INCREASES WITH EXERCISE
Usually decreases with Exercise

ECG Abnormalities



MINERAL DEFICIENCIES

COPPER DEFICIENCY

A : Anemia (MICROCYSTIC HYPOCHROMIC)
H : HAIR DEFECT
G : GROWTH RETARDATION
E : DEFECTIVE ELASTIN
O : OSTEOPOROSIS
M : MENTAL RETARDATION

CHROMIUM DEFICIENCY :

GLUCOSE INTOLERANCE

PHOSPHORUS DEFICIENCY :

RHABDOMYOLYSIS
HEMOLYSIS
ATAXIA
SEIZURES

ZINC DEFICIENCY

GROWTH RETARDATION
ALOPECIA
AGEUSIA
ANOSMIA
GONADAL DYSFUNCTION
DERMATITIS
IMMUNE DYSFUNCTION

MANGANESE DEFICIENCY

LIPID + CARBOHYDRATE METABOLISM ALTERED
GROWTH & SKELETAL DEVELOPMENTAL RETARDATION
UPPER BODY RASH

IRON DEFICIENCY

IMPAIRED COGNITION
POOR WORK PERFORMANCE
PREMATURE LABOUR
INCREASED PERINATAL DEATH

SELENIUM DEFICIENCY

SEEN IN KESHAN DISEASE
HYPOTHYROIDISM IN SUBHIMALAYAN ZONE

MOLYBDENUM DEFICIENCY

NEUROLOGICAL SYMPTOMS + OESOPHAGEAL CANCER

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TOXICITY/OVERDOSE

- COPPER TOXICITY : HEPATO TOXICITY
HEMOLYTIC ANEMIA
- BORON OVERDOSE : DEVELOPMENTAL DEFECT
MALE STERILITY
TESTICULAR ATROPHY
- IRON TOXICITY : INCREASED SUSCEPTIBILITY TO MALARIA
- MANGANESE OVERDOSE : PARKINSON LIKE FEATURES
- LEAD TOXICITY : GOUT LIKE FEATURES
∴ CALLED AS SATURNINE GOUT / LEAD NEPHROPATHY
HYPERURICEMIA, TUBULOINTERSTITIAL DISEASE
- CADMIUM TOXICITY : NEPHROTOXICITY with SEVERE BONE PAIN
COUGH COUGH NEPHROPATHY
- CHROMIUM TOXICITY : RENAL FAILURE + LUNG CANCER
↓
TUBULOINTERSTITIAL DS.
- ARSENIC TOXICITY : PERIPHERAL NEUROPATHY
GARLIC LIKE ODOUR
LUNG & NASAL CANCERS
- FLUORINE TOXICITY : CALCIFICATION OF TENDON/LIGAMENT
EXOSTOSES
- ZINC TOXICITY : PREDISPOSES TO PULMONARY FIBROSIS

ACID BASE REGULATION

✓ 70,00,000 nmol of H^+ produced by body per day

→ • Highly Cellulotoxic

• PLASMA CONC. H^+ = 40 nEq/ml
(This reduction is done by buffering)

CHEMICAL BUFFER SYSTEM

1. BICARBONATE BUFFER SYSTEM (BBS) MOST EFFICIENT
 2. PHOSPHATE BUFFER SYSTEM
- } EXTRACELLULAR BUFFER

- pK_a → Eg: H_2SO_4 $pK_a = 2.5$

↓
Dissociation constant

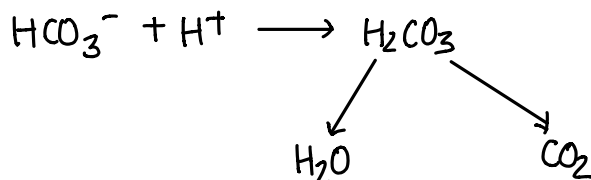
At this pH it will be
50% → Dissociated (H^+)
50% → Undissociated (AH)

• But Blood pH = 7.4

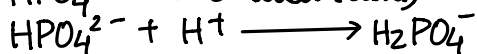
At this pH, H_2SO_4 → H^+ form ∴ can't be a buffer

∴ Buffer pK_a should be near to 7.4

- pK_a of HCO_3^- = 6.1 (weak acid) ∴ its concentration in Blood is high
IT CAN ACT AS BUFFER



- pK_a of HPO_4^{2-} = 6.8 (weak acid)



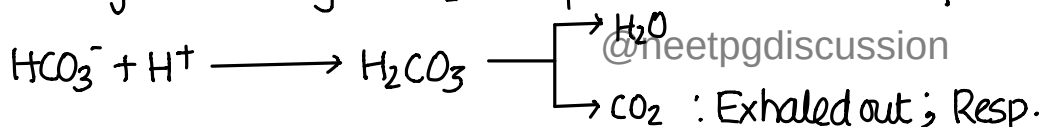
[BICARBONATE IS MORE EFFICIENT BECAUSE
 HCO_3^- CONC IS VERY HIGH THAN HPO_4^{2-}]

- INTRACELLUR BUFFER

1. PROTEINS : MOST IMPORTANT Hb
2. PHOSPHATE

RESPIRATORY BUFFER SYSTEM

- Exhailing or retaining of CO_2 in response to extracellular pH



NOTE:

Whenever there is Metabolic acidosis : Respiratory compensation is CO_2 being EXHALED OUT

Called as RESPIRATORY ALKALOSIS

RENAL BUFFER SYSTEM

- NON VOLATILE ACIDS PRODUCED/DAY : 80 mEq

↓
EXCRETED BY KIDNEY

- 4320 mEq of HCO_3^- FILTERED/DAY AT GLOMERULUS

↓
This all should be RE-ABSORBED
REABSORPTION OF HCO_3^- : 80% PCT
10% Thick Ascending Limb
10% DCT

For every molecule of HCO_3^- RE-ABSORBED, ONE H^+ IS EXCRETED.
∴ NEED 4320 mEq H^+

- Total H^+ Excreted/day $\longrightarrow 4320 + 80 = 4400 \text{ mEq}$
For excretion of 4400 mEq H^+ $\longrightarrow >2500 \text{ Liters of URINE/DAY}$

BUT CONCENTRATION OF H^+ in URINE $\longrightarrow 0.03 \text{ nEq/L}$

∴ Management of H^+ excretion done by urinary buffers

- (i) AMMONIA BUFFER
- (ii) PHOSPHATE BUFFER

NOTE :

- $\text{pK}_a = \text{pH}$ at which ASSOCIATED FORM = DISSOCIATED FORM
i.e. $\text{HA} = \text{A}$

- HANDERSON EQUATION :

$$\text{pH} = \text{pK}_a + \log \frac{\text{A}}{\text{HA}}$$

- BICARBONATE GENERATION = NET ACID EXCRETION IN URINE = 70-80 mEq/day
ACID is getting excreted in the form of : * Ammonium
* Titrable acids (Phosphate/
Creatinine/Butyrate)

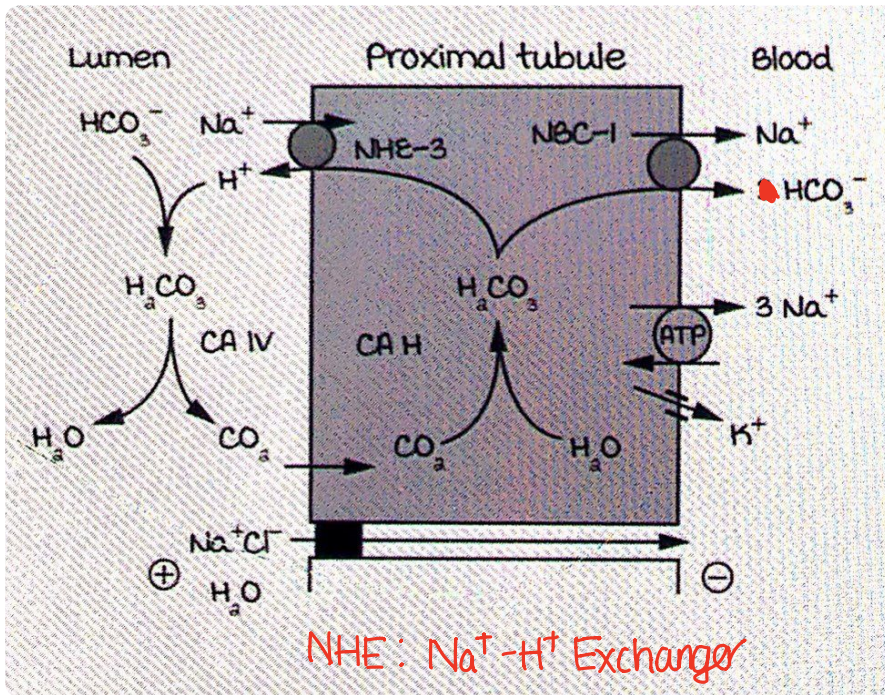
$$\therefore \text{NAE} = \text{NH}_4^+ + \text{Titration acid} = 70-80 \text{ mEq/day}$$

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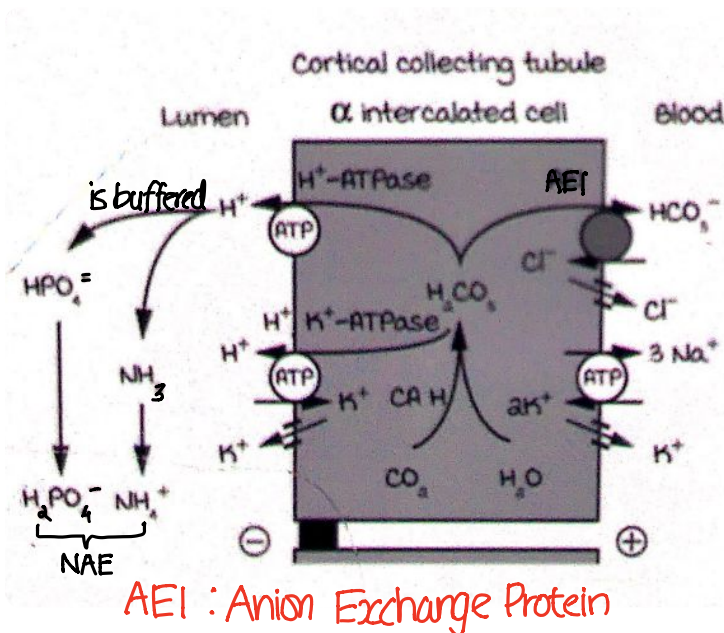
BICARBONATE AT PROXIMAL TUBULE



NOTE:

- EVERY MOLECULE OF HCO_3^- FILTERED INTO LUMEN OF TUBULE, ONE MOLECULE OF H^+ EXCRETED.
- PROBLEM IN NHE-3
↓
BICARBONATE EXCRETION
↓
∴ SEVERE ACIDOSIS
PROXIMAL RENAL TUBULAR ACIDOSIS

ACIDIFICATION OF URINE



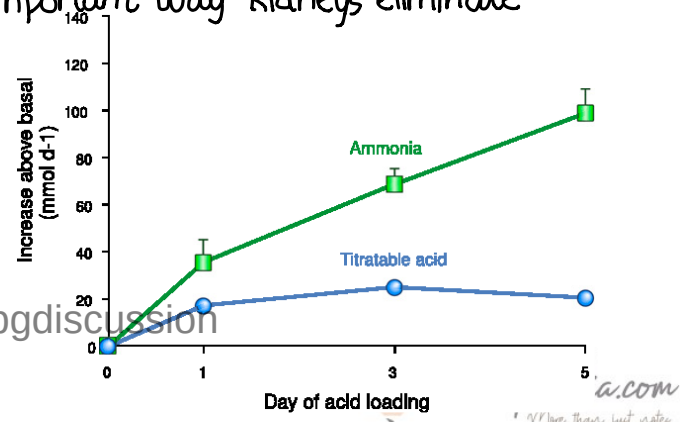
- ACIDIFYING MACHINERY → CORTICAL COLLECTING TUBULE (α INTERCALATED CELLS)
- Problem with H^+ - K^+ ATPase
↓
DISTAL TUBULAR ACIDOSIS (as H^+ can't excrete out)
- H^+ in urine is predominantly from α-intercalated cell

AMMONIA RECYCLING

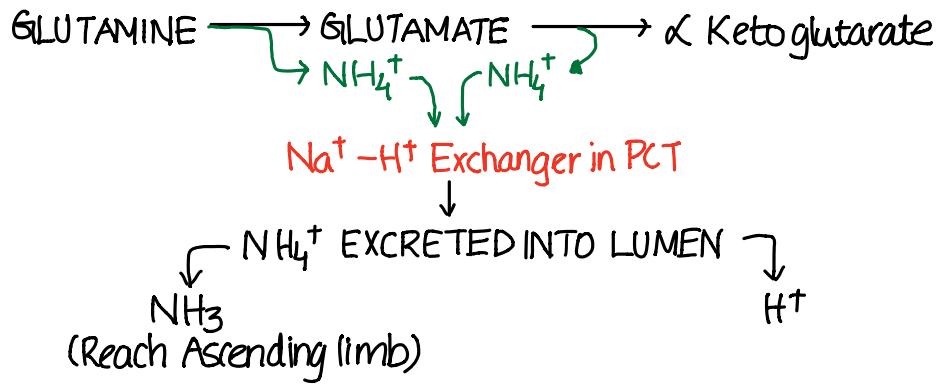
- Ammonium synthesis & excretion is the most important way kidneys eliminate non volatile acids.
- In Response to acid loading (From Graph)

- NOT TITRATABLE ACID INCREASE
- AMMONIUM EXCRETION INCREASE

∴ it's the Ammonium that is most important in acid load.



FORMATION OF AMMONIA: INSIDE PCT



- In response to $\uparrow$ acid load Kidney compensates by $\uparrow$ AMMONIA EXCRETION

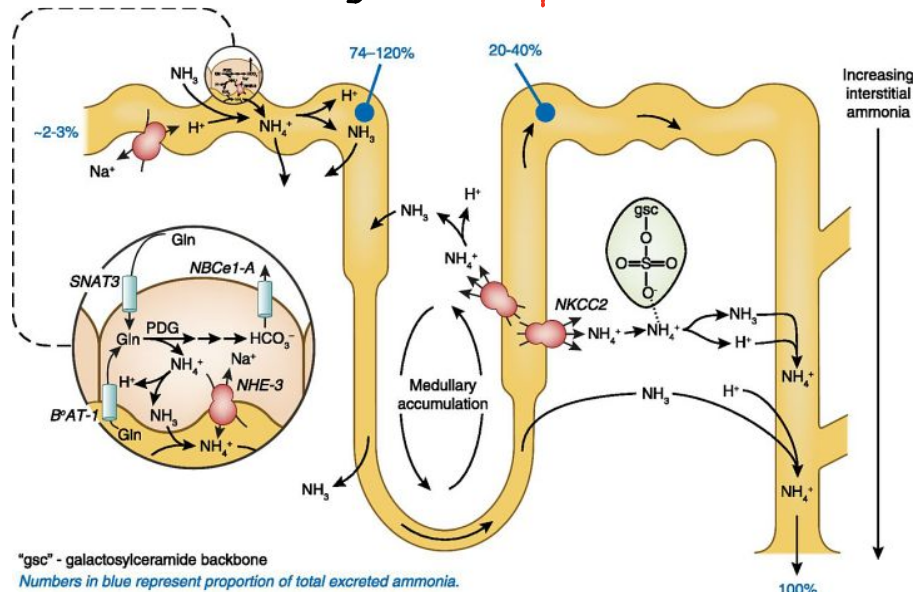
- STIMULI OF AMMONIA PRODUCTION $\rightarrow$

- METABOLIC ACIDOSIS
- HYOKALEMIA

- NH_3 at THICK ASCENDING LIMB $\rightarrow \text{Na}^+ \text{K}^+ 2\text{Cl}^- \rightarrow$ REACH THE INTERSTIUM

$\downarrow$
 $\text{NH}_4^+ \leftarrow \text{URINE} \leftarrow \text{COLLECTING DUCT}$
 - pKa of ammonia $\rightarrow 9.2 \therefore$ at PHYSIOLOGICAL pH 98% is NH_4^+

- AMMONIA generated in the kidney $\rightarrow \text{NH}_4^+$



ACIDS IN THE BODY

- 2 TYPES

(i) VOLATILE ACIDS

- Formed by metabolism of
 - carbohydrates
 - fats
 - proteins
- Excreted as CO_2 from lungs

(ii) NON VOLATILE ACIDS : (fixed acid) : 70-80 mEq/day = 1mEq/kg/day

- Formed by metabolism
 - Phospholipid
 - Nucleic acids

- Eg: Sulphuric acids, Phosphoric acid, organic acids
- Excreted by Kidneys

- Henderson Hasselbach's Equation : $pH = pK_a + \log \frac{[A^-]}{[HA]}$

- According to Henderson Hasselbach's Equation, important determinants of H^+
 - Bicarbonate (22-26 mEq/L)
 - pCO_2 (40mmHg)

First Defence	: Chemical Buffer
2nd Defence	: Respiratory Buffer
3rd Defence	: Renal Buffer

METABOLIC ACIDOSIS

- HEART :
 - ↓ myocardial contractility
 - Sympathetic overactivity
 - ↑ sensitivity to catecholamine
- CNS :
 - Lethargy, Disorientation, Stupor, Coma (HYPOACTIVITY)
- HYPERVENTILATION
- HYPERKALEMIA

- MEASURED CATIONS + UNMEASURED CATIONS = MEASURED ANIONS + UNMEASURED ANIONS

$$MC + UC = MA + UA$$

$$MC - MA = UA - UC$$

$$Na^+ - (Cl^- + HCO_3^-) = UA - UC$$

$$ANION GAP = UA - UC = 8-12 \text{ mEq/L}$$

$$\bullet \text{ CORRECTED ANION GAP} = \text{ANION GAP} + 2.5(4.5 - S. \text{Albumin})$$

CLASSIFICATION OF METABOLIC ACIDOSIS:

(i) HIGH ANION GAP MA: (Pneumonic : KUSMAL)

- KETOACIDOSIS → • DM

• Alcohol

• Starvation

→ Ethylene Glycol

} CAUSES FOR ENDOGENOUS KETOACIDOSIS

- Lactic acidosis

- Uremic acidosis

- Methanol/ethylene glycol

- Salicylate/Paraldehyde

} EXOGENOUS ACIDS

(ii) NORMAL ANION GAP MA/ HYPERCHLOREMIC METABOLIC ACIDOSIS

- Renal acidification defect → RTA (TYPE 1 or 2 or 4)

- GI Loss of HCO_3^- → • Diarrhoea

• Small bowel tumor

• Uretero sigmoidostomy

• Anion exchange resins



NOTE : $\text{ANION GAP} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$
 In ACIDOSIS $\rightarrow \downarrow \text{HCO}_3^- \rightarrow$ Compensated by $\text{Cl}^- \rightarrow \therefore \text{N ANION GAP}$
 But if Cl^- not compensates HCO_3^-
 $\downarrow$
 Some other molecule compensate
 $\downarrow$
 HIGH ANION GAP

METABOLIC ALKALOSIS

- HEART : Cardiac arrhythmias
 - BRAIN : Cerebral Insufficiency $\rightarrow$ HYPOXIA : Lt shift of ODC
 - NM SYSTEM : Muscle weakness
 - HYPOCALCEMIA
 - HYPOKALEMIA
 - HYPO MAGNESEMIA
- } $\therefore$ LOT OF EXCITABILITY ISSUES

CLASSIFICATION OF M. ALKALOSIS

(i) CHLORIDE/SALINE SENSITIVE/RESPONSIVE :

- * Urinary $\text{Cl}^- < 15 \text{ mEq/L}$ i.e URINE CHLORINE IS LOW
- * Vomiting
- * Nasogastric suctioning
- * Long term diuretic use
- * Cystic Fibrosis
- * Post hypercapnia
- * Pyloric Stenosis

(ii) CHLORIDE UNRESPONSIVE : SALINE RESISTANT

- * Urinary $\text{Cl}^- > 15 \text{ mEq/L}$
- * with HTN $\rightarrow$ RAS Activation
 - $\downarrow$ Aldosterone tumours
 - Liddle Syndrome
- * without HTN $\rightarrow$
 - Recent Diuretic Use
 - Barter syndrome
 - Gitelman syndrome

CHLORIDE DEPLETION

$\downarrow$
Contracted ECF volume

$\downarrow$
 $\oplus$ RAS & Aldosterone

$\downarrow$
 Na^+ Reabsorption
 $\text{H}^+ - \text{K}^+$ Excreted

$\therefore$ HYPOKALEMIA
ALKALOSIS

$\rightarrow$ More Na^+ Delivered distally
(Chloride get absorbed along with Na^+)

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ACID BASE REGULATION-2

ABG - ARTERIAL BLOOD GAS CONTRAINDICATION

- Abnormal Modified gas precautions
- Local infection / distorted anatomy at the puncture site
- Severe peripheral vascular disease of artery
- Active Raynaud's Syndrome

ABG - PROCEDURE AND PRECAUTIONS

- Preferred artery : • Radial artery : **MOST PREFERRED**
• Femoral artery
- **21 Gage** SYRINGE USED
- ① Flush the syringe with 0.5ml of 1:1000 Heparin
- ② Don't leave excess heparin in syringe

↑ HEPARIN → ↑ DILUTIONAL EFFECT → ↓ HCO_3^-
↓ PCO_2

- ③ Ensure no air bubbles

Air bubble = pO_2 mm Hg, pCO_2 0 mm Hg
∴ AIR BUBBLE + BLOOD = ↑ pO_2 , ↓ pCO_2

- ④ Early analysis & transported via cold chain

Change/10 min	Uniced 37°C	Iced 4°C
pH	0.01	0.001
pCO_2	1 mm Hg	0.1 mm Hg
pO_2	0.1%	0.01%

ESTIMATION OF H^+ FROM pH

- as pH ↓ → H^+ ↑

pH 7.4 → 40
pH 7.3 → 50
pH 7.2 → 62
pH 7.5 → 32

Estimating the H^+ ion from pH

7.0	7.1	7.2	7.3	7.4	7.5	7.6	7.7	7.8
			X 1.25		X 0.8			
100	80	62	50	40	32	25	20	16

⇒ Within narrow limits

0.01 change in pH $\approx$ 1 mEq / L change of $[\text{H}^+]$

7.40 = 40 7.39 = 41 7.38 = 42 7.37 = 43

NORMAL VALUES OF ABG

- pH → 7.36 - 7.44 (7.40)
- HCO_3^- → 24 - 26 mEq / L (24)
- pCO_2 → 35 - 45 (40)
- H^+ → 36 - 44 mEq / L (40 nEq / L)
- pH < 7.2 → Severe acidosis
- pH > 7.6 → Severe alkalosis

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CALCULATION OF BICARBONATE

STEP I : Obtain ABG & Serum Electrolytes simultaneously
(Validating the ABG)

STEP II : • $\text{HCO}_3^- / \text{MEASURED HCO}_3^- = 24 \times \frac{\text{PCO}_2}{\text{H}^+}$

• Measured HCO_3^- is compared with venous blood

• For ABG to be compatible

The measured HCO_3^- should be within ± 2 values compared to HCO_3^- of venous blood

EXAMPLE :

ABG :

pH : 7.3

$\therefore [\text{H}^+] = 50$

pCO₂ : 25

$$\text{HCO}_3^- = \frac{24 \times \text{PCO}_2 (25)}{\text{H}^+ (50)} = 12$$

→ Serum panel HCO_3^- : 11 $\therefore$ COMPATIBLE

STEP III : LOOK FOR pH

if pH > 7.4 → Alkalosis

if pH < 7.4 → Acidosis

if pH = 7.4 → Normal or Mixed ABG

STEP IV : Respiratory or metabolic component has changed in the direction of pH

EXAMPLE : I

pH : 7.32 ↓ → Acidosis

HCO_3^- : 16 ↓ → Acidosis

pCO₂ : 35 ↓ → Alkalosis

} METABOLIC ACIDOSIS

II

pH : 7.46 ↑ → Alkalosis

pCO₂ : 25 ↓ → Alkalosis

HCO_3^- : 20 ↓ → Acidosis

} RESPIRATORY ALKALOSIS

III :

pH : 7.42 : ALKALOSIS

HCO_3^- : 28 : ALKALOSIS

pCO₂ : 45 : ACIDOSIS

$\therefore$ METABOLIC ALKALOSIS

IV :

pH : 7.46 : Alkalosis

pCO₂ : 25 : Alkalosis

HCO_3^- : 16 : Acidosis

$\therefore$ Respiratory alkalosis

STEP IV : COMPENSATION

1. COMPENSATION FOR METABOLIC ACIDOSIS

- By Respiratory alkalosis
- $\text{HCO}_3^- \downarrow$ by $1 \text{ mEq/L} = \text{pCO}_2 \downarrow 1.2 \text{ mm Hg}$

EXAMPLE I : $\text{pH} : 7.10 : \text{ACIDOSIS}$
 $\text{pCO}_2 : 32 : \text{ALKALOSIS}$
 $\text{HCO}_3^- : 10 : \text{ACIDOSIS}$ } METABOLIC ACIDOSIS

Here HCO_3^- fall from $24 \rightarrow 10 = 14 \text{ mEq/L}$ fall

$$\therefore \text{COMPENSATION} = 14 \times 1.2$$
$$= 17$$

$$\therefore \text{The expected } \text{pCO}_2 = 40 - 17 = 23$$

$\therefore$ It's UNCOMPENSATED or PARTIALLY COMPENSATED METABOLIC ACIDOSIS
 $\therefore$ problem pCO_2 is 32 & the expected is 23

EXAMPLE II : $\text{pH} : 7.1 : \text{ACIDOSIS}$
 $\text{pCO}_2 : 32 : \text{ALKALOSIS}$
 $\text{HCO}_3^- : 12 : \text{ACIDOSIS}$
MET. ACIDOSIS
 $24 - 12 = 12 \text{ mEq/L}$
 $12 \times 1.2 = 14.4$
 $\therefore \text{Expected } \text{pCO}_2 = 40 - 14.4$
 $\cong 25$
 $\therefore$ UNCOMPENSATED/
MET. ACIDOSIS WITH MILD
RESP. ALKALOSIS

EXAMPLE III : $\text{pH} = 7.35 : \text{ACIDOSIS}$
 $\text{HCO}_3^- = 14 : \text{ACIDOSIS}$
 $\text{pCO}_2 = 28 : \text{ALKALOSIS}$
 $\therefore$ Metabolic acidosis
Fall = 10
 $\therefore$ expected fall in $\text{pCO}_2 = 10 \times 1.2 =$
 12
 $\therefore \text{expected } \text{pCO}_2 = 40 - 12$
 $= 28$
 $\therefore$ COMPENSATED/
SIMPLE METABOLIC ACIDOSIS/
MET. ACI with RESP. ALKALOSIS

EXAMPLE IV : $\text{pH} : 7.1 : \text{Acidosis}$
 $\text{pCO}_2 : 45 : \text{Acidosis}$
 $\text{HCO}_3^- : 10 : \text{Acidosis}$
METABOLIC + COMBINED ACIDOSIS

EXAMPLE V : $\text{pH} : 7.1 : \text{Acidosis}$
 $\text{pCO}_2 : 35 : \text{Alkalosis}$
 $\text{HCO}_3^- : 10 : \text{Acidosis}$
METABOLIC ACIDOSIS
Fall in $\text{HCO}_3^- = 14$
 $\therefore$ expected fall in $\text{pCO}_2 = 14 \times 1.2$
 $= 17$
 $\therefore \text{expected } \text{pCO}_2 = 40 - 17$
 $= 23$
 $\therefore$ UNCOMPENSATED

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ACID BASE REGULATION-3

COMPENSATION : STEP IV

2. COMPENSATION FOR METABOLIC ALKALOSIS

$$\text{HCO}_3^- \uparrow \text{ by } 1 \text{ mEq/L} = \text{pCO}_2 \uparrow \text{ by } 0.7 \text{ mmHg}$$

EXAMPLE 1 : pH = 7.45 : Alkalosis
 $\text{HCO}_3^- = 32$: Alkalosis
 $\text{pCO}_2 = 40$: Normal

∴ Metabolic Alkalosis

$$\text{Increase in } \text{HCO}_3^- = 32 - 24 = 8$$

$$\therefore \text{Expected } \uparrow \text{ in } \text{pCO}_2 = 8 \times 0.7 = 5.6$$

$$\therefore \text{Expected } \text{pCO}_2 = 40 + 5.6 = 45.6 \text{ mmHg}$$

∴ UNCOMPENSATED METABOLIC ALKALOSIS

NOTE :

pH = 7.30 : Acidosis
 $\text{HCO}_3^- = 14$: Acidosis

$$\text{pCO}_2 = 20$$

↓
 Met. Acidosis
 +
 Resp. alkalosis

$$\text{pCO}_2 = 28$$

↓
 Metabolic Acidosis
 Fully compensated
 or
 Simple metabolic
 acidosis

$$\text{pCO}_2 = 35$$

↓
 Metabolic acidosis
 Partially compensated

$$\text{pCO}_2 = 42$$

↓
 Met + Respiratory
 Acidosis

WINTERS FORMULA for METABOLIC ACIDOSIS

$$\text{pCO}_2 = 1.5 \times \text{HCO}_3^- + 8 \pm 2$$

EXAMPLE

pH = 7.3
 $\text{HCO}_3^- = 16$
 $\text{pCO}_2 = 30$

$$\text{pCO}_2 = 1.5 \times 16 + 8 \pm 2$$

$$= 32 \pm 2$$

$$= 30 - 34$$

3. COMPENSATION FOR RESPIRATORY ACIDOSIS & ALKALOSIS

- Respiratory

1 4 $\longrightarrow$ RESP. ACIDOSIS

2 4 $\longrightarrow$ RESP. ALKALOSIS

- RESP. ACIDOSIS

- Acute Resp. acidosis $\longrightarrow$ $pCO_2 \uparrow 10 \text{ mmHg}$ = $HCO_3^- \uparrow 1 \text{ mEq/L}$

- Chronic Resp. acidosis $\longrightarrow$ $pCO_2 \uparrow 10 \text{ mmHg}$ = $HCO_3^- \uparrow 4 \text{ mEq/L}$

EXAMPLE : Acute Resp. Acidosis

$pH = 7.25$	Acidosis	} Resp. acidosis
$pCO_2 = 55$	Resp. acidosis	
$HCO_3^- = 30$	Met. alkalosis	

$$pCO_2 \uparrow = 55 - 40 = 15$$

$\therefore$ for 10 $\uparrow \longrightarrow HCO_3^- \uparrow$ by 1

$$15 \uparrow \longrightarrow 1.5$$

$$\therefore HCO_3^- = 24 + 1.5 = 25.5$$

$\therefore$ It's Respiratory acidosis + Metabolic alkalosis
if $HCO_3^- = 24 \longrightarrow$ UNCOMPENSATED RESP ACIDOSIS

- RESP ALKALOSIS

• ACUTE RESP. ALKALOSIS $\longrightarrow$ $pCO_2 \downarrow 10 \text{ mmHg}$ = $HCO_3^- \downarrow 2 \text{ mEq/L}$

• CHRONIC RESP. ALKALOSIS $\longrightarrow$ $pCO_2 \downarrow 10 \text{ mmHg}$ = $HCO_3^- \downarrow 4 \text{ mEq/L}$

EXAMPLE : Acute

$pH = 7.46$	Alkalosis	} Respiratory alkalosis
$pCO_2 = 30$	Resp. Alkalosis	
$HCO_3^- = 22$	Met. Acidosis	

$$pCO_2 \downarrow = 10 \text{ mmHg}$$

$$\therefore \downarrow HCO_3^- = 2$$

$$\therefore HCO_3^- = 24 - 2 = 22$$

$\therefore$ COMPENSATED RESP. ALKALOSIS

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EXAMPLE :

A 40 yr old man is admitted for diarrhoea & breathlessness for 2 days
pH = 7.28 pCO₂ = 20 pO₂ = 100 HCO₃⁻ = 12
Na⁺ = 138 K⁺ = 2.0 Cl⁻ = 115 Urea = 70
S.Cr = 1.4 Sugar = 110

Ans : pH : 7.28 Acidosis
 pCO₂ : 20 Alkalosis
 HCO₃⁻ : 12 Acidosis } Metabolic acidosis

Fall is by 12, Expected fall = $12 \times 1.4 = 14.4$

∴ Expected pCO₂ = $40 - 14.4 \approx 25$

∴ Partly compensated

CALCULATION OF ANION GAP : STEP V

- Serum anion gap = $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) = 8 - 12 \text{ mEq/L}$
- Metabolic acidosis →
 - Loss of HCO₃⁻
 - If loss of HCO₃⁻ compensated by Cl⁻
Normal anion gap metabolic acidosis
- When loss of HCO₃⁻ is compensated by an ion other than Cl⁻ (Unmeasured anion) → High anion gap metabolic acidosis

EXAMPLE :

A 40 yr old man is admitted for diarrhoea for 1 week
& breathlessness for 1 day

pH = 7.3 pCO₂ = 30 pO₂ = 100 HCO₃⁻ = 16

Na⁺ = 140 K⁺ = 6.0 Cl⁻ = 102 Urea = 120

S.Cr = 5.4 Sugar = 110

Ans : → Here Metabolic acidosis

fall in HCO₃⁻ = 8

$8 \times 1.2 = 9.6$ fall in pCO₂

∴ compensated Met. acidosis

→ Anion gap = $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$
= $140 - (102 + 16)$
= 22 High Anion Gap
Met acidosis

→ K⁺ = 6.0 → Severe
Hyperkalemia

→ Creatinine high
∴ Renal failure

→ ∴ HAGMA + Renal Failure +
Hyperkalemia

Diagnosis : Uremic acidosis

- In High anion gap metabolic acidosis calculate delta ratio

$$\begin{aligned}\text{Delta Ratio} &= \frac{\text{Delta AG}}{\text{Delta HCO}_3^-} = \frac{\Delta \text{AG}}{\Delta \text{HCO}_3^-} \\ &= \frac{\text{AG} - 12}{24 - \text{HCO}_3^-}\end{aligned}$$

Normal = 1-2 → High anion gap Met. Acidosis

Delta Ratio $< 1 \rightarrow$ High anion gap Met. acidosis + Normal anion gap Met. acidosis

Delta Ratio $> 2 \rightarrow$ High anion gap Met. acidosis + Met. Alkalosis

EXAMPLE :

A 40 yr old man is admitted for diarrhoea for 1 week
& breathlessness for 1 day

$pH = 7.3$ $pCO_2 = 30$ $pO_2 = 100$ $HCO_3 = 16$

$$\text{Na}^+ = 140 \quad \text{K}^+ = 69 \quad \text{Cl}^- = 102 \quad \text{Urea} = 120$$

S.Gr = 5.4 Sugar = 110

→ Anion gap = $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$
 $= 140 - (102 + 16)$
 $= 22 = \text{High Anion Gap}$
 Met acidosis

$$\Delta AG = 22 - 12 = 10$$

$$\rightarrow \text{Delta Ratio} = \frac{22-12}{24-16} = \frac{10}{8} = \underline{1.2}$$

∴ Clear high anion gap
Metabolic acidosis

EXAMPLE

? A 45 yr old female is admitted for high fever & breathlessness, diagnosed to have acute Cholecystitis

$$pH = 7.3 \quad pCO_2 = 12 \quad pO_2 = 100 \quad HCO_3^- = 6$$

$$\text{Na}^+ = 140 \quad \text{K}^+ = 5 \quad \text{Cl}^- = 105 \quad \text{Urea} = 45$$

S.Cr = 1.6 Sugar = 120

Ans: → Metabolic acidosis

fall in $\text{HCO}_2^- = 18$

$$18 \times 1.2 = 20 = \downarrow p\text{CO}_2$$

$$p\text{CO}_2 = 20$$

$\therefore$ well compensated

∴ Met. Aci + Resp. alkalosis

→ Anion gap: $140 - (105 + 6) = 29$ (HAGMA)

$$\rightarrow \Delta \text{Ratio} = \frac{29-12}{24-6} = 17/18 < 1$$

∴ HAGMA + NAGMA +
Resp alkalosis

→ Sepsis & producing Lactic acidosis

ACID BASE REGULATION-4

URINE ANION GAP

- To differentiate normal AG Met. acidosis due to RTA or GI loss, urine AG done

$$\text{URINE AG} = \text{Urine Na}^+ + (\text{Urine K}^+ - \text{Urine Cl}^-)$$

Generally it's positive

Positive urine AG $\rightarrow$ RTA

Negative urine AG $\rightarrow$ GI loss of HCO_3^-

EXAMPLE

? A 50 yr old known COPD Pt, with cor pulmonale on
t/t with furosemide & admitted for severe vomiting.

$$\begin{array}{llll} \text{pH} = 7.41 & \text{pCO}_2 = 80 & \text{pO}_2 = 40 & \text{HCO}_3^- = 48 \\ \text{Na}^+ = 140 & \text{K}^+ = 4 & \text{Cl}^- = 105 & \text{Urea} = 45 \\ \text{S.Cr} = 1.2 & \text{Sugar} = 128 & & \end{array}$$

Ans: pH = 7.41 $\rightarrow$ put Alkalosis

$\therefore$ METABOLIC ALKALOSIS

Rise in HCO_3^- by = 24

exp. Rise in pCO_2 = 24×0.7
 ≈ 17

$\therefore$ Rise is = $40 + 17 = 57$

$\therefore$ RESPIRATORY ACIDOSIS

$\rightarrow$ COPD $\rightarrow$ Resp. acidosis

$\rightarrow$ Vomiting & diuretic use $\rightarrow$ Met. Alkalosis

EXAMPLE

? Based on the following information how would you describe the metabolic disorder

ABG: 7.24/42/99 100% O_2 sat on room air

Lytes: Na^+ 143 mEq/L, K^+ 4.0 mEq/L, Cl^- 100 mEq/L, HCO_3^- 21 mEq/L

Ans: $\rightarrow$ pH = 7.24 ACIDOSIS
 $\text{HCO}_3^- = 21$ ACIDOSIS
 $\text{pCO}_2 = 42$ ACIDOSIS } METABOLIC + RESPIRATORY ACID.

$$\rightarrow \text{Anion Gap} = 143 - (100 + 21)$$

$$= 22 \rightarrow \text{HAGMA} + \text{Respiratory Acid.}$$

$$\rightarrow \Delta \text{ Ratio} = \frac{22 - 12}{24 - 21} = \frac{10}{3} = > 3 \rightarrow \text{HAGMA} + \text{Resp acid} + \text{Met. Alkal.}$$



EXAMPLE

? A 10 year old teenager is found unconscious in his home, with an empty medicine bottle in hand. He is taken to ER, Based on following information what is the primary disturbance & does there appear to be appropriate compensation?

ABG: 7.21/42/90 100% O₂ saturation on air HCO₃⁻ 14 mEq/L

Ans: pH: 7.21
HCO₃⁻: 14
pCO₂: 42 } Metabolic acidosis
+ Respiratory acidosis

EXAMPLE

? A 10 year old pregnant insulin dependent diabetic patient was admitted with a h/o polyuria & thirst. She now felt ill & presented to hospital. There was h/o poor compliance with medical therapy.

• She was afebrile. Chest was clear. Circulation was adequate.

URINALYSIS : 2+ Ketones, 4+ glucose

• Na⁺ 136, K⁺ 4.8, Cl⁻ 101, pH = 7.26, pCO₂ 16 mmHg, pO₂ 128 mmHg, HCO₃⁻ 7.1 mmol/l

ANS: pH = 7.26 Acidosis
HCO₃⁻ = 7.1 Alkalosis
pCO₂ = 16 Acidosis } Metabolic Acidosis

fall in HCO₃⁻ = 17
expected pCO₂ = 40 - (17 x 1.2)
= 20 } Resp. alkalosis

AG = 136 - (101 + 7)
= 28 } HAGMA (Due to Diabetic Ketoacidosis)

$\Delta \text{Ratio} = \frac{28-12}{24-7} = \frac{16}{17} = < 1$ } HAGMA + NAGMA

↓
Due to RTA due to Diabetes
(not clear)

EXAMPLE

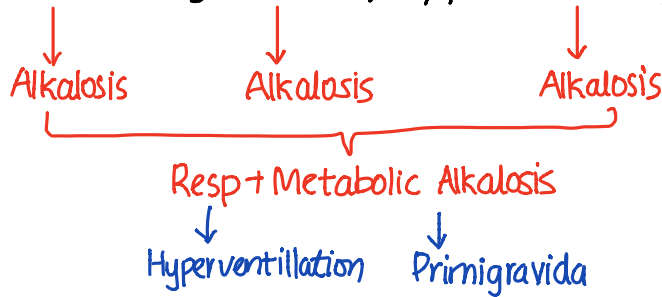
? A 60yr old woman was admitted with lobar pneumonia. She was on thiazide diuretic for 0 months following a previous admission with congestive cardiac failure. The ABG:

K⁺: 2.1, pH: 7.64, pCO₂: 32 mmHg, pO₂: 75 mmHg, HCO₃⁻: 33 mmol/l

↓ Alkalosis
↓ Alkalosis
↓ Alkalosis
∴ Metabolic + Resp. Alkalosis @neetpgdiscussion
Chronic thiazide use → Metab. Alkal.

EXAMPLE

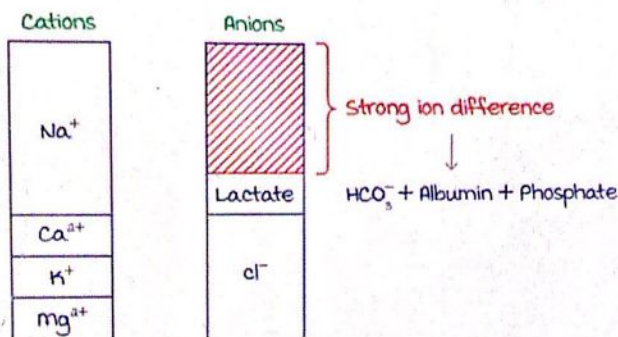
? 21 year old Primigravida with hyperemesis.
 $\text{pH} = 7.55$, $\text{HCO}_3^- = 30 \text{ mmol/L}$, $\text{pCO}_2 = 25 \text{ mmHg}$



STRONG ION DIFFERENCE

$$\text{STRONG ION DIFFERENCE} = (\text{Na}^+ + \text{K}^+ + \text{Mg}^{2+}) - (\text{Cl}^- + \text{Lactate}^-)$$

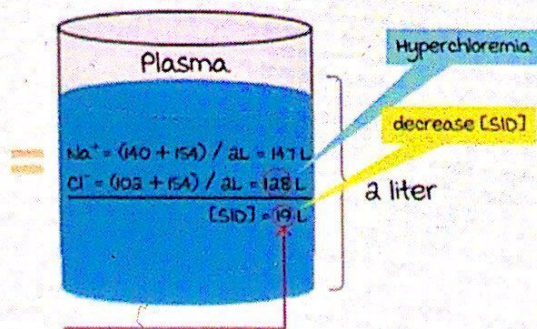
- Difference between measured strong cations & anions
- NORMAL SID $\rightarrow 40 \text{ mEq/L}$



$$\text{SID} = \text{HCO}_3^- + \text{Albumin} + \text{phosphate}$$

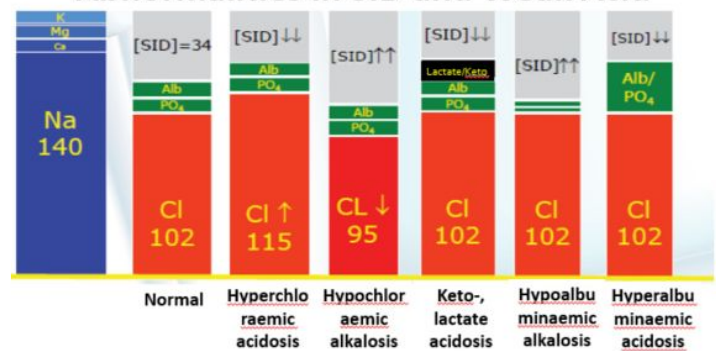
- MET. ALKALOSIS $\rightarrow \text{SID} \uparrow$
- MET. ACIDOSIS $\rightarrow \text{SID} \downarrow$
- DEHYDRATION $\rightarrow \text{SID} \uparrow$
- LACTIC ACIDOSIS $\rightarrow \text{SID} \downarrow$
- HYPO ALBUMINEMIA $\rightarrow \text{SID} \downarrow$

Saline cause acidosis by decreasing [SID] due to the hyperchloremic



$[\text{SID}] = 19 \downarrow \rightarrow \text{pH} \downarrow \rightarrow \text{more acidosis}$

Abnormalities in SID and Weak Acid



NOTE : Adding Ringer Lactate to plasma

$$\text{Na}^+ = (140 + 137) / 2 = 138$$

$$\text{Cl}^- = (102 + 100) / 2 = 101$$

$$\text{after adding Ringer SID} = 37$$

$$\text{initially} = 140 - 102 = 38$$

$\rightarrow$ Cause acidosis
 But less than that of saline

NOTE: Base excess of -2 to +2 is normal
 -2, -3, -4 etc → Met. acidosis
 > +2 → Met. alkalosis

$$SID = 40 \text{ mEq/L}$$

Whatever it decreases from 40 is Base excess negative : MET. ACIDOSIS
 Whatever it increases from 40 is Base excess positive : MET. ALKALOSIS

OSMOLARITY

- Calculated osmolarity = $2\text{Na}^+ + \frac{\text{B. Glucose}}{18} + \frac{\text{BUN}}{2.8} = 285 - 290$

- Measured osmolarity by Osmometer

$$\text{OSMOLAL GAP} = \text{Measured OSMOLARITY} - \text{Calculated Osmolarity} = > 10$$

- in
- Methanol poisoning
 - Ethylene Glycol poisoning
- Diagnosis : High anion gap metabolic acidosis + metabolic alkalosis + Respiratory acidosis

PROBLEM-1

? A young women presented with a h/o Watery diarrhea two day after railway journey. She is febrile, tachycardia, has dry mouth & cold extremities. Her BP was 90/60 mmHg.

INVESTIGATIONS

Blood Urea	: 40 mg/dl
S. Creatinine	: 1.4 mg/dl
S. Na^+	: 135 mmol/L
S. K^+	: 2.2 mmol/L
S. Cl^-	: 108 mmol/L
S. HCO_3^-	: 18 mmol/L
Anion gap	: 0 mmol/L
Albumin	: 2.5 g/dL
Corrected AG	: $0 + 2 \times (4.5 - 2.5) = 13$

ABG ANALYSIS

pH	: 7.31
pCO_2	: 3.5 mmHg
PO_2	: 95 on room air
HCO_3^-	: 16 mmol/L
$\text{O}_2 \text{ sat}$	: 95%

Ans: • pH: 7.31 Acidosis
 HCO_3^- : 18 Acidosis
 pCO_2 : 3.5 Alkalosis } 1° disorder Metabolic acidosis

- pCO_2 expected = $40 - (6 \times 1.2) = 33 \rightarrow$ Respiratory alkalosis
- AG = 0 : Normal ; Corrected AG = 13 : HAGMA
- A Ratio = $\frac{14 - 12}{24 - 18} = \frac{2}{6} = \leq 1 \rightarrow$ HAGMA + NAGMA + Resp alkalosis

BP 90/60, Shock, Lactic acidosis ↓ Diarrhoea ↓ Hyperventilation

PROBLEM-II

? An insulin dependent diabetic patient stopped taking insulin for 2 days & presented to casualty with vomiting & breathlessness. He complains of severe thirst. On examination his BP was 100/70 mm Hg, pulse rate 110/min regular & had acidotic breathing. Systolic examination was otherwise unremarkable. Clinical diagnosis: Patient has diabetic ketoacidosis

INVESTIGATIONS

Blood Urea	: 300 mg/dl
S. Creatinine	: 1.4 mg/dl
S. Na^+	: 135 mmol/L
S. K^+	: 5 mmol/L
S. Cl^-	: 100 mmol/L
S. HCO_3^-	: 12 mmol/L
Anion gap	: 23 mmol/L
Albumin	: 3.5 g/dL
Corrected AG	: 25
Urine Ketones	: positive

ABG ANALYSIS

pH	: 7.25
pCO_2	: 27 mmHg
PO_2	: 81 on room air
HCO_3^-	: 12 mmol/L
O_2 sat	: 95%

Ans : HAGMA + Compensated Metabolic acidosis

$$\Delta \text{ Ratio} = \frac{25-12}{24-12} = \frac{13}{12} = 1.2 \rightarrow \text{HAGMA}$$

No indication to give HCO_3^-

NOTE :

if < 7.1 plan for correction of HCO_3^-
Bicarbonate deficit = $[24 - \text{HCO}_3^-] \times 0.6 \times \text{Body weight}$

1 amp of HCO_3^- contain = 10ml

1ml = 1.7 mEq

10ml = 17 mEq

50% of deficit should correct in 24 hrs

PROBLEM-III

? A 65 year old woman who has been diabetic & HTN for 5 years on oral hypoglycemic agents & indapamide developed cellulitis of the foot following injury 15 days prior to admission. She was admitted with complaints of muscle weakness. On examination patient was hypovolemic BP 110/70 mm Hg, Hypotonia, with grade 4 muscle power in all four limbs.

INVESTIGATIONS

Blood Urea	: 360 mg/dl
S. Creatinine	: 1.2 mg/dl
S. Na^+	: 132 mmol/L
S. K^+	: 2.6 mmol/L
S. Cl^-	: 82 mmol/L
S. HCO_3^-	: 18 mmol/L
Anion gap	: 32 mmol/L
Albumin	: 4.5 g/dL

ABG ANALYSIS

pH	: 7.41
pCO_2	: 32 mmHg
PO_2	: 71 on room air
HCO_3^-	: 20 mmol/L
O_2 sat	: 95%

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Corrected AG : 32



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Ans: Respiratory alkalosis (Indapemide - diuretic)
 expected $\text{HCO}_3^- = 22$: Met-acidosis

AG: HAGMA (Hypovolemia & Sepsis)

$$\Delta \text{Ratio} = \frac{32-12}{24-18} = \frac{20}{6} = >2 : \text{Met. alkalosis also}$$

PROBLEM IV

? A 75 yr old man with a longstanding h/o heavy smoking is brought to hospital in a febrile & confused state. He rapidly becomes drowsy. SpO_2 is 80%. Clinical assessment indicates the patient is likely to have chronic resp-acidosis. He is hypoxic, so there is possibility of lactic acidosis in addition.

INVESTIGATIONS

Blood Urea : 360 mg/dl
 S. Creatinine : 1.2 mg/dl
 S. Na^+ : 132 mmol/L
 S. K^+ : 2.6 mmol/L
 S. Cl^- : 82 mmol/L
 S. HCO_3^- : 18 mmol/L

ABG ANALYSIS

pH : 7.21
 PCO_2 : 70 mmHg
 PO_2 : 55 on room air
 HCO_3^- : 26 mmol/L
 $\text{O}_2 \text{ sat}$: 80%

Ans: Respiratory + Metabolic acidosis
 AG: HAGMA (Sepsis & Lactic acidosis)
 $\Delta \text{Ratio} = \frac{20}{6} = >2 : \text{HAGMA} + \text{Met. alkalosis}$

↓
 due to smoking

↓
 Cause not clear

PROBLEM V

? A 50 yr old man was admitted with a 3 day h/o abdominal pain & vomiting. The patient was pale & tachypneic, BP was not recordable, & abdomen was rigid. He was resuscitated with IV fluids & taken up for emergency laprotomy which revealed infected ascitic fluid & perforated duodenal ulcers.

INVESTIGATIONS

WBC : 14500/mm³
 Hb : 14g
 Blood Sugar : 102 mg/dl
 Creatinine : 0.8 mg/dl
 Na : 120 mmol/L
 K : 4.4 mmol/L
 Cl : 84 mmol/L
 HCO_3^- : 15 mmol/L
 Case histories : 257

ABG ANALYSIS

pH : 7.21
 PCO_2 : 30 mmHg
 PO_2 : 85% on room air
 HCO_3^- : 14.6 mmol/L
 $\text{O}_2 \text{ sat}$: 80%

Ans: Metabolic acidosis + Resp. component
 • HAGMA
 • $\Delta \text{Ratio} : 2 : \text{Metabolic alkalosis}$
 HAGMA

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◉ A 60 year old lady is admitted with complaints of anorexia and diarrhea. Significant past illness included type II DM, hypertension, chronic osteomyelitis and recurrent urinary tract infection. O/E volume status- Dehydrated, BP 110/80mm Hg, no evidence of active osteomyelitis

Investigations(On Admission)

Parameter	On Admission	After 48hours	Parameter	On Admission	After 48hours
RBS	410 mg	167 mg	pH	7.193	7.267
Urea	218	199	PCO ₂	25.6	31.4
Creatinine	4.3	4.2	HCO ₃ ⁻	9.6	14
Na	119	131	PaCO ₂	109 mm Hg	114.7
K ⁺	7.2	4.8	O ₂ sat	93%	95%
Cl ⁻	96	98			
AG	13	19			
Albumin	2.5g	3g			
Corrected AG	16	22			

Solve this

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